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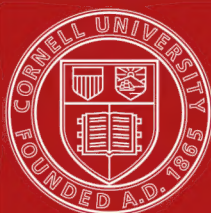
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**B. V. S.**

**THIS BOOK BELONGS**  
**.....TO THE.....**  
**Department of Agriculture,**  
**STATE OF NEW YORK,**

**THE PRINCIPLES AND PRACTICE OF**  
**VETERINARY MEDICINE**





# THE PRINCIPLES AND PRACTICE OF VETERINARY MEDICINE

BY

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NINTH EDITION

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## PREFACE TO THE NINTH EDITION.

THE rapid strides that have recently been made in veterinary medicine make it expedient, and indeed necessary, to issue a new edition of this work. Requests and applications from many quarters, both at home and abroad, and the suggestions of numerous friends have also had considerable influence upon my decision to reorganise the work, and reconstruct many of the chapters which have, since the issue of the 1897 edition, become out of date.

In the present edition special attention has been given to the modern methods of sero-therapy and vaccine-therapy, and their applicability to veterinary medicine as revealed by the discoveries in the field of bacteriology. The distinction that has been drawn between bacteriology and protozoology is now a vital one, and is, indeed, all-important. By the former is meant the study of those diseases that are caused by schizomycetic or bacterial organisms; the latter, which opens up an entirely new field of observation, is the study of animal parasites, such as trypanosomata, piroplasmata, and others of the same sort. Considerably more space than formerly has also been given in this edition to parasitology, which is becoming more and more intimately connected with medicine; by this I mean the study of those parasites *other* than bacteria and protozoa which infest the systems of domestic animals.

I have found it necessary, under present-day conditions of specialisation, to ask for the co-operation of other investigators



and I have been so fortunate as to obtain the valued help of, amongst others, Major Baldrey, who has aided me in reconstructing the chapters on infectious diseases and parasitology. My most sincere thanks are also due to Sir John MacFadyean, whom I have found myself quoting continually. So great is my obligation to him that, in despair of recording every point, I have instructed the friend who has framed my index to add the Latin words *et passim* (which may be freely translated, "on every other page") to his name, as it has been added to Gamgee, Dick and Nocard. I must further acknowledge my great indebtedness to my colleagues in the University of Liverpool, and especially to Professor Ronald Ross, Dr. Breinl, Professor Sherrington, Mr. Newstead, Mr. Matheson, Mr. S. E. Walker, Dr. Stephens, of the Tropical School, and many members of my profession. Finally, both Major Baldrey and myself most sincerely thank Mrs. Baldrey for her great assistance, and Mr. C. D. Campbell, of Edinburgh, for so carefully and skilfully compiling the index and tables of contents, in addition to much other help, as also my son, E. Owen Williams, for much assistance in revising proofs.

I have, for the reasons stated above, every confidence in issuing this revised edition (the ninth) of my late father's *Veterinary Medicine* to the veterinary profession and to all interested in the subject. I have the liveliest recollections of the kindly welcome which was accorded to the previous efforts of my father and myself, and I earnestly hope that the present volume may meet the same cordial reception.

W. O. W.

THE UNIVERSITY, LIVERPOOL,  
May, 1909.

P.S.—I feel that a short postscript is necessary as a sort of obituary tribute to a distinguished French veterinary surgeon.

I had the good fortune, when I was a student, to spend some months at the college at Alfort, near Paris, and there to come under the influence of M. Edouard Nocard, a veterinarian known over the whole civilised world by his writings on scientific subjects, and one who has influenced, directly and indirectly, the lives and careers of thousands of students. M. Nocard was present on May 11, 1903, at the Conference of Tropical Medicine held in Liverpool, and made a brilliant speech, in which, after praising the efforts made to advance the study of tropical diseases and tropical medicine, he pointed out that there was a distinct lacuna in the educational arrangements, and that was the institution of a school of veterinary medicine. M. Nocard's speech will be found on page 15 of Vol. V. (new series) of the Thomson, Yates and Johnston Laboratories Report, 1903, and is deserving of the most careful study. In a startling way he shows us how vitally important the study of the tropical diseases of animals is for us—a nation with great colonial dependencies—and narrates a particular instance which came under his own notice. I have no doubt that this speech gave the final impulse to Sir Rupert Boyce—whom M. Nocard frequently mentions in terms of admiration, and almost of affection—and led to the establishment, under his statesmanlike assistance, of the School of Veterinary Medicine in this University. It had always been my ambition to obtain University education for veterinary students, and Sir Rupert having his ambition, and I mine, running on parallel lines, a veterinary school was inaugurated in this University, as M. Nocard had so ardently desired.

W. O. W.





## PREFACE TO THE EIGHTH EDITION.

SINCE the last edition of this work appeared, in 1893, many important discoveries have been made in Pathological and Biological Science, more particularly in the departments that treat of the evolution, the physiological or pathological effects, and all the various phenomena associated with the development and life-history of the lower forms of life known as Bacilli, Bacteria, and Microbes. The Author has therefore been compelled to completely recast the Bacteriological portions of his work.

In 1882 an attempt was made by certain parties to disparage and minimise the importance of a discovery which the Author claimed to have made—namely, the organism of the Tick. Since then the Author has continued assiduously his investigations, with the result that his former conclusions have been abundantly confirmed. For further information on this point, he refers the reader to the chapter on what is popularly termed Louping-*Ill*, which he has ventured to term *Ixodic Toxæmia*. This chapter, along with that on Texas Fever (termed by the Author *Ixodic Anæmia*), which he had special opportunities of studying while in Jamaica in the summer of 1896, will, he trusts, be found interesting as being founded on the theory that certain saprophyte filaments, innocuous in themselves, may, when ingested with foods which they infest, become pathogenic or disease-inducing to the higher animals, when they have

passed through the blood of the lower forms of life, such as Ticks, and have been transmitted by these into the circulation of the higher animals. This view to some extent supports that of Buchner, who states that the ordinary Hay Bacillus—*Bacillus subtilis*—attains all the virulent properties of the *Bacillus anthracis* after it has been cultivated in animal media, meat-extract, blood, &c.

Since this work has gone to press, the Author has received further corroboration of the seemingly anomalous occurrence of simple filaments as retrogressive stages in the development of an organism, apparently dependent upon a change in the nutrient medium—a fact previously noticed in the course of his “Louping-ill” investigations, 1882, which has led to a considerable amount of controversy.

These so-called “involution” stages, as exhibited by the *Bacillus typhosus*, will be found described and illustrated in a paper by Dr. Edward P. Carter in the *Bulletin of the Johns Hopkins Hospital* for June of this year.

The thanks of the Author are specially due to his colleague, Dr. JAMES HUNTER, whose unwearied labours and whose acute judgment and profound learning have contributed greatly to the fulness and accuracy of many of the illustrations, in themselves unique, in the present work.

W. W.

NEW VETERINARY COLLEGE,  
EDINBURGH, *September*, 1897.

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THE  
PRINCIPLES AND PRACTICE  
OF  
VETERINARY MEDICINE.

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CHAPTER I.  
INTRODUCTORY.

THE external diseases incidental to the domesticated animals having been discussed in my work on Veterinary Surgery, I purpose in the present volume to enter into a consideration of the more purely medical or internal ailments from which our patients are liable to suffer.

I have advisedly made a distinction between Veterinary Medicine and Veterinary Surgery, not only for the reason that the subjects, thus divided, are more easily dealt with, but because conclusions which may be considered almost hypothetical and speculative in medicine, are replaced in surgery by the tangible and demonstrable.

Medicine is studied as a science and as an art: as a science, when it inquires into all the circumstances under which diseases become developed, the condition of their existence, and into their nature and causes; as an art, when it is directed towards the recognition, the prevention, and cure of diseases. In fact it is the art of understanding the nature of diseases, so far as to appreciate their causes, to prevent their occurrence when possible, and to promote their cure or to relieve them when they occur.—(BIGLOW. AITKEN.)

In order that the student be enabled to comprehend Medicine as a science, and its application as an art, it is necessary he

should understand **PATHOLOGY**, which, in its full and proper meaning, implies a knowledge of diseased processes, abnormal conditions, and morbid structures, as well as what precedes them and what results from them. For this purpose a knowledge of many collateral branches of science is essential, more particularly a knowledge of **PHYSIOLOGY**; and no one can be a physiologist without being an anatomist and a chemist. By **Physiology** is meant that science which treats of the conditions, phenomena, and laws of life whilst the animal body is in a state of health. Without a knowledge of the laws of health, it is impossible to grasp and comprehend the laws of disease, for it may be truly said that the latter are but perversions of the former, and are natural, or physiological, under the operation of existing circumstances and causes.

In addition to **Pathology** and **Physiology**, **Medicine** comprehends **THERAPEUTICS**, or the science which explains the actions of remedies upon the animal body, the means by which disease may be naturally overcome, and a return to health assisted and promoted; and **HYGIENE** or **PROPHYLAXIS**, which treats of the sanitary condition, food, and surroundings whereby disease may be prevented, and all other methods by which health may be preserved.

Disease may also be studied **CLINICALLY**: that is to say, disease may be studied as it presents itself in each particular case to the attention of the observer. The term *clinic* can scarcely, with propriety, be applied to any method by which diseases of the lower animals are studied, as it means "a patient who keeps his bed," but for the want of a better, and as it is now a generic term, I am constrained to retain it.

Before proceeding further with our object, it is necessary that I endeavour to give a definition of disease, and this I can only do by following the rules already laid down by our latest pathologists. A definition of disease can only be arrived at by comparing it with the standard of health, and health, says **Williams**, "consists in a natural and proper condition and proportion in the functions and structures of the several parts of which the body is composed;" but no fixed rule can be applied to this, for what is health in one may be disease in another, and there are degrees or gradations of health which cannot be said to be due to disorder or disease. For example, one animal may fatten and maintain the most robust health upon the same

quantity of food that would keep another thin and poor. "In plain words, health does not signify any fixed and immutable conditions of the body, nor does health necessarily imply the integrity of all the bodily organs: it is not incompatible with great and permanent alterations, nor even with the loss of parts, that are not vital. Our comprehension of health being thus indefinite, our idea of disease must be indefinite also; and the best definition that can be given of it is, that it is a deviation from the state of health, consisting generally in a change in the properties or structure of any tissue or organ, which renders such tissue or organ inadequate to the performance of its healthy actions and functions."—(WATSON.)

It must not, however, be supposed that disease, as exhibited by an unnatural or morbid condition, and by phenomena which are seemingly abnormal, is unnatural in itself; for in reality disease may be looked upon as the natural expression of a combination of conditions, the essential and proper consequences of some cause or influence which has acted, or is acting, upon the animal body. To adduce a familiar example, let us suppose a blister is applied to the skin: the inflammation of the skin which is thus produced is certainly unnatural, and may with propriety be called disease; but if we look further into the matter, we can easily understand that the inflammation, vesication, or even ulceration so induced are the natural results of the action of the irritant,—in fact, the proper and healthy reaction of a healthy organism to the irritation of the blister. The absence of this reaction—the skin remaining healthy—under such conditions would indeed be unnatural, unlooked for, and incomprehensible. This may be taken as a type of cause and effect in all diseases: it is not the inflamed condition of the skin, nor even the constitutional disturbance which may be caused by a severe blister, but the presence of the blister that is really unnatural. This illustration, homely as it may seem, is important, as all real advancement in prevention and treatment is based upon a due appreciation of the causes of disease; for in the past, more particularly in Veterinary Medicine, the aim and purpose of practice has been directed to the treatment of symptoms—to deal with effect, often ignoring the cause. Hence the many systems, founded on erroneous bases, by which diseases have been combated, have been productive of much harm.

## CHAPTER II.

### PATHOLOGY.

**PATHOLOGY**, or more properly, when applied to the lower animals, **Zoo-Pathology**, is derived from the Greek words Πάθος, a disease, and Λόγος, a discourse—the doctrine of disease; and is divided into **GENERAL** and **SPECIAL PATHOLOGY**.

**GENERAL PATHOLOGY** includes—*1st.* **ETIOLOGY**, or a knowledge of the causes of disease: *2nd.* **SEMIOLOGY** or **SYMPTOMATOLOGY**, or a knowledge of the morbid phenomena or symptoms by which disease is manifested: *3rd.* **PATHOGENY**, which deals with the seats or localities of disease: *4th.* **NOSOLOGY**—its division and classification: *5th.* **DIAGNOSIS**—the methods by which it is detected—its distinction: *6th.* **PROGNOSIS**—its probable results; *7th.* **MORBID ANATOMY** and **MORBID HISTOLOGY**—the methods by which the morbid alterations of structure and the elementary constituents of diseased products are discovered.

**ETIOLOGY.**—*The causes of disease*, or, in other words, the circumstances which precede it, and to which its occurrence is due, are arranged under three heads, namely—the *predisposing*, the *exciting*, and the *proximate*.

The term *proximate* is used to represent the pathological condition or essential bodily change on which the symptoms of disease depend; in fact, the proximate cause has been stated to be the disease itself, and for this reason some writers have expunged the term, and have been content to arrange the causes of disease under the heads *predisposing* and *exciting*. Later writers, however, have revived the term, and Dr. Bristowe gives the following illustration: “A woman who has been frequently exposed to the contagion of scarlet fever without taking the disease, becomes at the period of childbirth again exposed, and now suffers from a virulent attack. Here, parturition (which,

as we know, renders women peculiarly susceptible of the contagious fevers) is the predisposing cause, the scarlatinal organism is the exciting cause, and the inflammatory processes going on in the skin, tonsils, and elsewhere, the proximate causes of most of the symptoms which the patient manifests. But the exciting cause of the scarlet fever is obviously the proximate cause of that disease, and the proximate causes of its several secondary phenomena are just as obviously their exciting causes." The distinction between the exciting and the proximate causes is thus purely artificial; whereas the differences between the predisposing and exciting causes are generally well marked; but the co-operation of both of these kinds of causes is, however, generally necessary to produce disease.

*Predisposing causes* of disease differ from a *predisposition* to disease. The first may be certain influences operating upon the animal body from without, such as heat, cold, vitiated atmosphere, inordinate work, the quality of the food, poisons, &c.; whilst a *predisposition* to disease is always *intrinsic*, existing within the animal body, and is very frequently found to arise from some hereditary taint. For example, horses of certain breeds become roarers, or otherwise defective in their wind, from no appreciable cause. Animals thus affected are said to have a hereditary predisposition to these infirmities. In such instances, the predisposition may be truly said to be the predisposing influence which has given rise to the disease of which roaring or other defect in the respiratory function is the symptom; but it cannot be maintained that a violent inflammation of the larynx or of the lungs, when succeeded by roaring, constitutes a *predisposition*, although they are most certainly the predisposing, and in some instances the exciting causes of the same pathological condition.

Predisposing causes of disease commonly consist of various circumstances which influence the functions or structures of the body in an unfavourable manner, yet short of actual disease; or, in other words, those general, non-specific conditions which by their influence so alter the health of the system, or the condition of parts of it, as to render them specially suitable for the development of certain diseases, provided that an animal so predisposed be subjected to the influence of the excitants of such diseases.

*The exciting causes* of disease are those circumstances and

agents which, operating on the body, especially when predisposed, may excite disease; amongst them are included those specific causes, or elements of disease, which stamp their individuality on the morbid processes which ensue in the animal body when the germs of such diseases happen to become implanted therein (as in glanders, rabies, rinderpest, scabies, &c.), and are divided into the cognizable and non-cognizable. The first includes all the physical and other agents of whose existence we can take cognizance, independently of their operation in producing disease; but these so-called non-cognizable causes are now found to consist of certain organisms, the majority of which have been demonstrated to be living matters having specific effects.

The predisposing and exciting causes of disease, when existing within the system, are called intrinsic, *endopathic*, or internal; but when they arise without the system, they are denominated extrinsic, *exopathic*, or external causes of disease.

*Predisposing causes of diseases.*—The most important and generally recognised predisposing causes of disease are—

(1.) *The influence of age.*—This is not so striking in the lower animals as in man, but still it plays an important part. In the dog, for example, the period of dentition renders the animal liable to fits of convulsion, paralysis, disturbances of the digestive process, with vomition, irregularity of the fæcal discharges, weakness, and even inflammation of the eyes, and attendant unthriftiness. Rickets is also a disease which is only seen during the early period of life, and is witnessed in all our domesticated animals, but more particularly in the dog.

Canine distemper may be manifested during any period of life: as a great rule, it is only seen during the first few weeks or months of the animal's existence. Again, the *strangles* of the horse is generally a disease of adolescence.

It is also well known that the invasion of parasites is much more common during the earlier period of an animal's life; thus we find the *Cœnurus cerebralis* developed in the brains of sheep and cattle during the first months of life. The *Strongylus filaria*,<sup>1</sup> of the lamb, and its analogue, the *Strongylus micrurus* of the calf, as a general rule induce disease in these animals in early life. The *Trichonema arcuata*,<sup>1</sup> witnessed by me in Icelandic ponies, and referred to in the latter part of this work, are rarely found in horses above two years old.

<sup>1</sup> *Trichonema arcuata* is the larval form of *Schlerostomum tetracanthum*.



The bones are also much more liable to disease during youth than middle age ; for example, osteo-porosis is rarely witnessed in an animal which has passed its fifth year. I say rarely witnessed, for I have seen some horses affected with this disease which had passed the age of seven years ; but even here I was induced to think that the morbid changes had probably originated during the earlier years of the affected animals' lives.

Again, we find that disease of the facial bones is generally seen in young horses, and is doubtless closely connected with the process of dentition. Nor must we forget to mention that dentition is not always free from attendant ill consequences ; in some cases the crowns of the temporary molars are not shed, but become entangled in the newly cut permanent ones, causing difficulty in mastication, indigestion, and unthriftiness, more particularly in horned cattle ; whilst in the horse, at about the age of four years, a true dental cough results from irritation induced by the cutting of the sixth molar tooth.

It becomes a matter of speculation whether those causes of lameness which are so commonly met with in young horses are results of predispositions or not. When it is taken into consideration that the bones of the young are more vascular or succulent, contain less earthy matter than those which have arrived at full maturity, and that these young bones are subjected to inordinate work, concussion, and alteration of incidence by erroneous shoeing, we must conclude that there is not always a predisposition to disease, but that disease is induced by subjecting the animal to work which its physiological condition and strength are unable to withstand. At the same time, many animals develop diseases of the bones and joints without the aid of exciting causes, which leads us to conclude that in them there is at this period of life a predisposition to disease.

Both foals, calves, and lambs are very early—indeed often within the first few days of their existence—subject to disease of their articulations, very frequently without any apparent cause. Here we may safely conclude that great vascularity of the extremities of the bones constitutes a predisposing cause of disease.

During the middle period of life, animals as a rule have few special tendencies to disease. Such affections as navicular arthritis, and those conditions of the respiratory tract which give rise to *roaring*, *whistling*, &c., are more frequently witnessed during the

middle period ; but as the predispositions to these affections are supposed to be hereditary, they will receive further consideration.

The cow is perhaps an exception to that exemption from diseases occurring in middle life, for in this animal we find that when its powers are fully developed, liability to that fatal disease, parturient apoplexy, is much enhanced.

At the approach of old age, and during its continuance, diseases arising from tumours, degeneration of organs and tissues, manifest themselves ; the heart, liver, &c. undergo fatty metamorphosis ; the circulation becomes feeble ; digestion impaired ; the blood-vessels, particularly those of the brain, undergo a calcareous change ; the joints become stiff ; the bones brittle, and the cutaneous surface liable to be infected by various parasites. In the dog, the crystalline lens undergoes a retrograde change, and soft cataracts are commonly seen ; whilst deafness and dropsy are not rare amongst dogs and cats of advanced age.

In the ox tribe, again, we find that age influences predisposition, as the disease *black-leg* chiefly affects animals under eighteen months old, and the disease *anthrax* affects mature animals, and in the dog it is usually the young that have *distemper*.

*Sex.*—The difference in the organisation of the male and female. In addition to those variations in diseases necessarily arising from the peculiarities of the generative organs of the two sexes, as affections of the testicles, &c., in the one, and of the ovaries or uterus, as well as hysteria, in the other, we find that sex has to a limited extent an influence at least in one disease, which occurs in both sexes ; thus roaring is much more frequently met with in horses and geldings than in mares.

*Peculiarities of breed and conformation.*—In connection with breed and conformation, we often witness predispositions to certain forms of disease ; as, for example, canker and chronic grease, common enough in heavy-legged cart-horses, are but rarely seen in the better bred ones. Roaring is very often associated with certain conformation of the neck. Large horses with long necks, particularly if fine or small at the throat, are much more predisposed to roaring than those with shorter necks ; smaller horses are more rarely affected, and ponies very seldom indeed become roarers. Round-chested horses are more liable to become broken-winded.

There are also certain forms of lameness which conformation and size have much to do with. Calcification of the lateral cartilages is rarely witnessed in the better bred, but is very common in the cart-horse. Navicular disease, so rife amongst better bred horses, is a very rare cause of lameness in the cart-horse; and, not to adduce too many examples, high-bred, nervous animals are more liable to nervous diseases than those of a lower breed.

*The effects of colour* in predisposing to disease are very curious, as shown in the frequency of melanotic neoplasms in grey or white horses; and the liability to cutaneous eruptions upon the white parts of the body only, has caused one disease to be called "white face and foot disease."—(*Crusta labialis*, see *Veterinary Surgery*.) It is also well known that pure white dogs and cats often are either born deaf or become so at an early period.

There is also a uterine affection which is said to occur only in white heifers, but this is open to doubt.

Further, there is a prejudice against horses which are known as "washy" chestnuts, as they are regarded as generally unthrifty and liable to lung and stomach disorders.

The action of the sun in the tropics is particularly noticeable on white-nosed horses. The white muzzle becomes sunburnt and painful, while the remainder of the animal's body is unaffected.

*Tribe or species.*—The influence of species in favouring or resisting certain forms of disease is really remarkable. Thus the horse, whilst liable to glanders, resists diseases to which cattle and sheep are peculiarly liable, such as rinderpest, pleuropneumonia, eczema epizootica. Rabies, again, is primarily a purely canine disease. Charbonous disease is transmissible to man and to all the domesticated animals, with the exception of the domestic fowls and birds generally; but here it has been found—and probably this discovery explains why some contagia affect certain animals only—that resistance to charbonous contagion is due, not to conformation, but to temperature; for when the temperature of the fowl has been lowered by removing its feathers and keeping it in water, it has become affected with the disease.

*Temperament*, which consists in excess of or defect in some

function or set of functions, certainly predisposes to particular diseases.

The *sanguine* temperament, implying an activity of the circulatory and blood-producing organs, tends to diseases of an inflammatory character. This temperament is best exhibited in highly-bred horses, whereas in lower-bred animals the lymphatic temperament seems to predominate. In this temperament there is a deficiency of arterial tonicity, a want of nervous power, the circulation is languid, the capillaries and lymphatics, particularly in the extremities and depending parts of the body, are often congested and the limbs œdematous, which conditions disappear for a time with exercise. Inflammatory diseases run a less favourable course in animals with this temperament, there being a tendency to the effusion of serum, to deliquescence of organs, and to gangrene, and not to the exudation of plastic lymph, as in animals in which tonicity is good.

Cattle and sheep are generally considered to possess a lymphatic temperament. This supposition is to my mind hypothetical, for it is found that these animals do not exhibit the same tendency to œdematous swellings and other signs of debilitated circulatory powers as the horse, and, in the ox particularly, lymph is abundantly exuded in some inflammations.

The *nervous* temperament is manifested by agitation and excitement, and although it is called nervous, it does not follow that an animal possessing it has extra nervous power. Indeed, the contrary is often the case, for an animal of this kind generally becomes exhausted in a very short time. The term seems to imply a want of proportion of some of the functions or properties of the nervous system.

Nervous animals are easily frightened, and a sudden fright may be followed by serious illness, such as violent purgings, loss of appetite, tremblings and excitement, which may continue for several hours or even days, and, what is more serious, sudden death from rupture of the heart. I have seen one sudden death in the horse from this cause—of one that was quite well at the time. In another, not of death, but of serious illness. I have it recorded that an old hunter, every time it was shod, was seized with purging, loss of appetite, tremblings, snorting with fear, and other symptoms of excitement, which always continued for three days.

*Previous diseases* are also predisposing causes of new ones. Thus we find many horses recovering or recovered from an epizootic become the subject of rheumatism in a joint, bursa, or tendon. Catarrhal fever is often succeeded by *purpura*. Chorea succeeds distemper in the dog. Lymphangitis predisposes to succeeding attacks of the same affection; and if under this head we classify artificial diseases, as induced by the operation of medicine, purgative, or other evacuant, we can understand how, during the prevalence of an epizootic, the disease is easily excited in an animal which has been physicked a short time previously. Even a surgical operation will, during the prevalence of an epizootic disease, assist its development in an animal previously healthy. In fact, anything which will tend to lower the animal's resistance—or, as Wright puts it, anything that reduces the opsonic power—will predispose the animal body to an attack of a bacterial disease, as during the menstrual period, when it has been found that the opsonic content of the blood is always low (*vide* chapter on “Opsonins”).

*Present disease, and conditions bordering on disease.*—Diseases of the heart, causing engorgement of the veins, often lead to congestion of the lungs and liver, dropsy of the cavities and areolar tissue; excessive evacuations, as in diarrhœa and diabetes, predispose to other diseases, as glanders or tubercle; feeble digestive and assimilative powers, to anæmia and dropsies; inflammations, particularly in horned cattle, to caseous tumours, in parts other than those originally inflamed.

*Hereditary tendency.*—Many diseases, such as roaring, curbs, spavin, ringbones, navicular disease, chorea or stringhalt, are more common in certain breeds of horses; tuberculosis in the best breeds of horned cattle, etc.

Dr. Fleming states that French authors are unanimous in asserting that the disease termed “roaring” in the equine species, and which now generally affects horses in Normandy, was unknown there until the arrival of Danish stallions.

The influence of climate in overcoming hereditary predisposition is well shown in India and South America, where horses the progeny of roarers are as a rule exempt from this infirmity.

*Diathesis, idiosyncrasy, or aptitude.*—Diathesis is a term used

to describe a particular tendency to certain forms of disease, such as the rheumatic, tubercular, and scrofulous. In animals of this constitution, the particular disease to which they are predisposed, or to which they have a tendency, is caused by different exciting circumstances, and serious diseases are induced by trivial causes, although such animals may present no external signs of idiosyncrasy.

*Exopathic predisposing causes.*—Whilst the endopathic causes of disease are generally beyond the influence of our preventive power, those existing external to the animal are to a very great extent subject to our control, and, by a careful study of them, we discover that many diseases are preventable. As a rule, however, extrinsic causes are generally excitants of disease, and it would be a needless repetition if I were to describe them here. I shall therefore pass on to—

*The exciting causes of disease.*—These again present themselves to us under two heads—namely, the intrinsic or endopathic, and the extrinsic or exopathic. The endopathic exciting causes are—(1.) *Mechanical*, which include strictures of orifices and tubes, by contraction of their coats, pressure from without, or impacted concretions—as stricture, intus-susception, intestinal or urinary calculi; presence of parasites in the ducts of the liver, in the bronchial tubes and cerebral hemispheres; the pressure of tumours on the brain and other parts of the body; herniæ; obstructions in the larynx or œsophagus; nasal polypi, &c.; and (2.) *Chemical causes*, which include all retained secretions of excretory organs—such as urea and the other products of the metamorphosis of the nitrogenous constituents of the food and body—occurring as a result of disease of the kidneys, or when nitrogenous products are too abundantly present in the blood, as in azoturia. Again, when from some impediment to the respiratory function carbonic acid is retained, the animal may die from delirium and coma. If the liver fails to perform its function, jaundice follows, succeeded by anæmia, dropsy, and death.

Chemical and mechanical causes are also external or exopathic, and include all poisonous substances, mineral and vegetable—as acids, caustic alkalies, opium, strychnia, aconite, lead, &c. &c., and all forms of external violence.

*Disease Germs.*—Contagia, morbid poisons, and specific disease virus are now included under exopathic causes of disease,

although the evidence is not very clear upon all points, but they are generally propagated by infection, contagion, and inoculation (see "Origin of Contagious and Infectious Diseases").

Exopathic predisposing and exciting causes are those due to the influence of climate, food and water, ventilation and drainage, locality, geological formation, weather and seasons, work, and the want of it.

Of the *influence of climate*, the diseases known generally as tropical diseases, which are the result of specific infections particular to any zone or region in which the specific organism can only live, or where its propagation is carried on through an intermediate parasitic host incapable of surviving under any other climatic conditions, afford well-known examples.

*Food and water.*—The solid and liquid ingesta are fertile sources of disease—*1st*, by being insufficient or ill-proportioned in *quality*; *2d*, by being deficient or excessive in *quantity*.

Unsuitability of food, either in consequence of a deficiency, over-abundance, or improper combination of nutritive constituents, is a very frequent cause of disease. This has been well shown in the experiments of Majendie and others. Dogs, geese, donkeys, and other animals, when fed entirely on sugar, gum, starch, oil, or butter, died with symptoms of starvation, almost as soon as if they had been kept without food. Even bread, when too fine, is insufficient nourishment. A dog fed on pure white bread lived only fifty days, whereas another fed with the coarsest brown bread was well nourished, and seemed capable of living for an indefinite period. Again, according to the researches of a Commission of the French Institute, animals fed on pure fibrin or albumen died of starvation, almost as soon as if not fed at all.

Experiments have proved that in order to support health and strength it is essential that, in addition to water, food contain at least three classes of constituents, namely—(*1st.*) Nitrogenous, to nourish muscular and other albuminoid tissues: (*2d.*) Hydrocarbons and carbo-hydrates, which undergo combustion in the body, and assist in the maintenance of animal heat and in the assimilation of the nitrogenous compounds; and (*3d.*) Salines, to supply materials for the building up of the solid structures of the body, maintaining them in health, and assisting in the processes of assimilation and elimination, conveying new materials

into the system and removing old ones out of it. If these various constituents are deficient, absent, or present in undue quantities, health cannot be maintained, and common experience has taught that all animals are kept in the best health when fed on a mixed diet. And as types of all proper food we have two examples. (1st.) Milk offers us the best example, as it contains casein—nitrogenous, oil and sugar—hydrocarbons, water and salts. Hence young animals thrive best, and are maintained in health by the food which nature has provided for them. (2d.) Grass may also be adduced as a food containing exclusively all the ingredients required for the support of animal life.

As examples of the bad effects of food good in itself, but ill-proportioned in quality, we may adduce those diseases, namely, fatty degeneration of the liver and anæmia, so commonly met with in sheep too exclusively fed on turnips. During good seasons, when the turnips are firm, well-grown, and healthy, they tend to overload the system with saccharine matter, and to induce a degenerative change in the liver, which renders it of a palish yellow colour, friable, and incapable of performing its functions. This condition may be associated with fatness: indeed sheep so affected begin to die when almost fit for the butcher, and the best plan, when they thus begin to fall off, is to make the remainder of the flock so fed, into mutton as soon as possible.

During wet seasons, turnips, although apparently fully grown, may contain but little nourishment, but are loaded with watery particles. Animals then have to consume large quantities to maintain life, and in consequence the digestive powers become weakened; debility, anæmia, and death are the results.

Now if the stockowner bear in mind that the food with which he is supplying his animals does not contain the essential constituents of sound food, he will avoid his losses by supplementing turnips with cake, corn, and particularly with some long food, as it must be borne in mind that all ruminants thrive best upon material which requires remastication. The horse too, though not a ruminating animal, does not thrive except on food, some of which at least is coarse, and requiring much mastication; and the bad effects of feeding the horse on a diet easily swallowed are seen when it is fed on cooked food or on bran mash exclusively. I have seen cases



of fatal impaction and of rupture of the stomach caused by feeding on bran alone; and my experience points to numbers of instances where severe indigestion, with colicky pains, and fœtor of the breath, have been induced when coarse food has been withheld from horses suffering perhaps from a sore throat or other disease.

But whilst hay or straw, which may be called the coarser articles of diet, are necessary, they are insufficient alone to maintain an animal in robust health, as the indigestibility of the quantities necessarily ingested becomes a source of disease, indigestion, broken wind, languor and debility, or lead to such a condition of the system as to predispose it to succumb to the influences of epizootics.

In addition to being insufficient or ill-proportioned, food may be *bad* in quality, as in rainy seasons, where the vegetation is too watery in its nature, its nutritive constituents washed out as it were; or damaged by mould, or other causes in operation and extending over districts and even countries. What is more common after a bad harvest, hay or corn, than to see scores of horses affected with the disease termed diabetes insipidus, induced by some as yet unknown agent developed in the food by the operation of wet, heating, and fermentation, or all of these operations combined?

Again, it is found that wheat and rye are affected with ergot, and oats with a fungus growth, eminently fatal in its action on the animal body; and that all kinds of forage by becoming rusty acquire unhealthy properties, infected with cryptogamic plants belonging to *Uredo* or *Puccinia* species, or mouldy—blue-mould—when attacked by the *Mucor mucedo*.—(See FLEMING'S *Sanitary Science and Police* on this point.)

Of the bad effects of grass in rainy seasons, when it is loaded with watery particles, numerous instances of tympanitis, diarrhoea, and dysentery of a fatal nature, particularly among sheep, are witnessed during wet years.

But if the season be too dry, forage becomes hard, innutritious, and indigestible from want of moisture as one of its constituents; causing constipation, impactions, with their attendant conditions of the body, unthriftiness and debility, leading on to anæmia, and even death. In young cattle particularly, this condition of the pastures, at first giving rise to indigestion,

induces, if long continued, a mal-condition of the osseous system, whereby the bones becomes fragile or brittle, with stiffness of the joints and liability to spontaneous fractures. In some parts of Scotland a similar condition of the skeleton is brought about by feeding young cattle on turnips grown on land highly dressed with the phosphates, and urinary calculi are not uncommon in animals highly fed on cakes and other artificial food.

Food may be excessive or deficient in quantity. The more common effects of food taken in excessive quantities are laminitis, congestion of the brain, and enteric affections. As a rule, we find that animals partake of food in quantities sufficient to satisfy appetite and maintain health; but there are exceptions to this, and we find some, particularly horses, habitually greedy in their desire for food, eating voraciously, hurriedly, and masticating imperfectly. Others again are voracious from accidental long fasting, and the evil consequences of this kind of feeding are a source of great loss to the horseowner, and of great anxiety to the veterinary surgeon, as most of the fatal cases of disease of the digestive organs arise from this cause. To avoid such loss, a little forethought would go a long way. If a horse be habitually greedy, it should be made to take the edge off its appetite by an allowance of hay before its corn, then be fed sparingly on the latter, and the quantity which is generally given at once ought to be divided into two or three parts and given at intervals; if this be not done, colic, rupture, enteritis, or laminitis may ensue. If a horse seem voracious from long fasting, a similar course would often avert an attack of perhaps a fatal disease; and at all times, if it be deemed expedient, owing to a damaged condition of the grain, to give cooked food—an unnatural kind of food for horses, cattle, and sheep—it ought to be given in small quantities, and often.—(See Diseases of the Digestive Organs, also effects of Potatoes.)

Food defective in quantity or nourishment causes debility, wasting, cedematous legs, susceptibility to the attacks of parasites, anæmia, dropsy, and even death.

Chusatz found that in animals gradually starved to death the temperature of the body progressively declined, and unless maintained artificially, the animals seemed to die of cold. All

the textures, even the bones, sustained great loss of weight, but those of the nervous system less than any others.

Sudden change of food, even if it be of good quality, is often a cause of disease. For example, what is more common than a sudden outbreak of disease amongst cattle, particularly young ones, hardly kept in the winter, when first turned into rich pastures. The same applies to sheep, and I have often witnessed a fatal form of enteric disease amongst sheep—in one instance extending to cattle and horses—when depastured during early summer upon land lately drained and limed. So great was the fatality, that the farm seemed unfit for grazing purposes, for, after liming and draining had been completed, the more benefit the land seemed to derive from the cultivation and the richer the pasture, the greater the mortality.

*The influence of water.*—Much prejudice exists, particularly among horsemen, as to the use of water. In consequence of this we find that hunters and racehorses are severely punished before they are called upon to hunt or race, by having but a very limited and insufficient allowance of water prior to the performance of their task. What harm a sufficient supply of water, partaken of several hours before the hunt or race, can effect, is beyond my comprehension; indeed I have found that a hunter properly fed and watered on a hunting morning has been enabled, provided always that it is otherwise in condition, to do its work with greater ease, and to last longer than animals deprived of so necessary an element as water.

The fetish of withholding a sufficiency of water would appear to be a survival of what is to the present day carried out by some of the tribes of Central Asia, in which neighbourhood the horse was first known. The men are keen horsemen, and very fond of horse-racing. Their races are usually four to six or eight miles, and their method of preparing the horse is to keep it on a minimum of water, and no soft food, so much so that the horse's skin is very badly "hide-bound." It is wonderful, however, how fresh and well the horses will pull up after a trying gallop of six miles, being ridden hard every furlong of the way.

Furthermore, it would appear that a considerable number of cases of lameness in the horse are attributable to a deficient

supply of drinking water, and in consequence the kidneys are rendered unable to eliminate the salts from the blood, and these salts become deposited in the neighbourhood of joints, and give rise to rheumatic and gouty affections of them. In my own practice I have frequently treated such cases systemically as well as locally, and have had most beneficial results.

Water containing organic impurities, or too hard from the over-abundance of earthy salts—that is to say, water containing more than twelve degrees of hardness—is apt to induce disease.

Organic impurities, either suspended or in solution, and more particularly during the hotter months of summer, are apt to induce diarrhoea, anthracoid and septic diseases, which may prove fatal. Animals certainly seem to become habituated to impure water, and sometimes prefer it to that which is clean and wholesome, but even these are not exempt from its evil consequences; and indeed the very depravity which leads them to prefer what is so unnatural may be looked upon as of itself a diseased condition. It may, however, be stated that there seems to be a resistance, particularly in cattle, to the ill-effects of water contaminated with vegetable products, whereas, if these be of animal origin—human ordure, decomposed blood, milk, &c.—disease is almost certain to follow. Continual drinking of very warm water is said to induce disease.

Pure water may with benefit and advantage be allowed in such quantities as the animal seems to require, taking its condition into consideration at the time. If it be heated or exhausted by work, water must be supplied in moderate and repeated quantities, and not too cold, until thirst is assuaged, otherwise enteritis, diseases of the skin, or inflammation of the feet, may result. With this exception, I repeat that water ought at all times to be freely but judiciously allowed.

Water in the form of dew or hoar frost is believed to be a cause of disease. It is very possible, if a very hungry animal were to eat dew-covered grasses ravenously and quickly, that digestive disturbances might arise; but I fail to see how food, masticated and retained in the mouth until its temperature is elevated almost as high as that of the body, can be inductive of disease. Dr. Fleming tells us (*Sanitary Science and Police*)

that honey-dew has been supposed to cause aphthous and other affections. "Intelligent agriculturists in Saxony have remarked this dew as a cause of epizootics, and the shepherds take the precaution of leaving their crook on the grass in driving home their flocks, and examining it in the morning before driving them out again. If they observe the dew which has gathered on the crook to be only water, the flocks may then be allowed to pasture; but if it resembles oil or honey, then they must remain until the dew has evaporated."

This is an old superstition, which has probably as much value as the ancient idea that miasma or mist in the evening was the cause of malaria. The mosquito and mist usually arise together from the swampy places, and of course the mist was blamed. We are continually learning that the spread of disease is more and more closely associated with intermediary parasites, and it is highly probable that a heavily dew-laden grass encourages the presence of some biting, disease-spreading insect.

*Geological formation and locality.*—Mr. Robertson, Kelso, in his little work *Hints to Stockowners*, says—"The substrata on which soils rest, and to which in part they owe their formation, is always an important element in determining their character, and one which remains undisturbed in its permanence, notwithstanding cultivation, and the improvements of modern scientific agriculture. From an acquaintance with the nature of this formation, we can in many cases predict what will be the character of the stock bred and reared on such lands, as also the diseases to which they are more particularly liable, or from which they are exempt. For example, it is a fact well known to the majority of our sheep-breeders, that on certain soils, chiefly those resting on the igneous rocks, sheep are liable to suffer from a form of abdominal consumption, known as *pinning*, and that no system of treatment is so efficacious as their removal to soils resting on the sandstone formation. There are also diseases of particular structures of the animal body, as the bones and nervous system—*enzootic*—that is, confined to circumscribed districts of country, which seem to owe their origin to the redundancy or absence in the soil, and materials grown thereon, of certain organic or inorganic materials. These diseases are always difficult of prevention, when only methods of cultivation, or

systems of folding the animals on these lands, are adopted. They would require to have access to those situations known to be dangerous only at particular periods, and to have what food is given them from such soils supplemented by others of a very different nature."

In several parts of Scotland, it is well known that stock suffer to an enormous extent in certain pastures during spring, or until the appearance of the white clover. It is difficult to account for this, unless by the supposition that, owing to the geological formation of such districts, the grasses, no matter how abundant they might be in quantity, are defective in some constituents, and calculated by their deficiency in some necessary nutritive material to render the animal organism particularly susceptible to any infectious disease that may become prevalent, or to render the animal susceptible to attack, in a pathogenic sense, of an organism which under ordinary circumstances would be practically harmless, this susceptibility remaining until such grasses have attained a certain age, coincident with the period of the blossoming of the white clover.

In soils deficient in certain mineral constituents, the plants grown thereon will also be deficient in those, if not artificially supplied; as, for example, as stated by Mr. Fleming, there is no phosphate of lime in the soil of the alluvial tracts on the banks of the Rhine. What is necessary for the growths of plants is derived from the atmosphere, which supplies no less than 400 grammes to each hectare,<sup>1</sup> according to Burrell, and which is deposited by the rain. In dry years, however, this supply fails, and the plants are then deficient in this most essential ingredient. The consequence is, that the creatures which consume these plants suffer more or less, and this is now recognised as one of the causes of that special affection of the bones of animals in those regions which has received the name of osseous cachexia.

It is well known in Scotland that certain nervous diseases, more particularly that known as "loup-ill" in lambs, are only witnessed in certain localities—very often the breadth of a river being sufficient to separate the unhealthy from the healthy ground. Many sheep-farmers and shepherds have long believed, what is now demonstrated to be the case, that

<sup>1</sup> A hectare is about  $2\frac{1}{2}$  acres.

“loup-ill” is only seen upon land infested with ticks, and that ticks are the cause of the disease; while it is possible to have land infested with ticks, and yet have the sheep free from loup-ill, as all ticks do not seem to contain disease germs, and it is clearly proved that loup-ill is not seen where ticks are absent.—(See “Loup-ill.”)

The influence of marshes and undrained lands in predisposing to and exciting disease is well known, and need but be merely referred to here. Such diseases as rheumatism, dysentery, and typhoid diseases in animals, and intermittent fevers in man, are traced to these water-logged areas, which are so favourable to the growth of organisms, or to their reproduction either directly or through the intermediary host of one of the many biting insects which inhabit such damp places, and act as a syringe does to the experimental pathologist, in spreading the germs of disease to man and animals. It is now demonstrated that many diseases are due to specific germs, and that they cannot be developed except by the introduction of the germs into the animal body. The subject will be discussed hereafter: at the present it is only necessary to state that moist lands have a great influence on the development of germs, and that since this country has been to a great extent drained, at least some, such as malaria, no longer exist, and many others, such as anthrax, are in a great measure mitigated. Again, rot in sheep, as well as other parasitic diseases, prevail to a much greater extent on damp than on dry soils, and the influence of a wet season, even on dry and well-drained soils, induces the development of many diseases, amongst which the grouse disease may be cited.

*Imperfect ventilation*, to which may be added defective drainage, is a fertile cause of disease; indeed it may be stated that defective ventilation is a source of greater loss to owners of horses than all other causes of disease combined, particularly in large towns. A deficiency of oxygen, excess of carbonic acid, and a scanty supply of fresh air, stint the vital processes, and the gradual accumulation of the ammoniacal products of the decomposition of excrementitious matters by which animals are surrounded, are almost certain excitants to diseases, particularly those of the respiratory organs; and when disease is once excited by such causes, a recovery becomes almost an impossibility until the

sufferer is removed from their influence. It will be pointed out hereafter that in the treatment of lung inflammation, pure air is an absolute necessity; and if we bear in mind that impure air is often a cause of this inflammation, can we wonder at the fatality of diseases when animals suffering from such a disease are kept under the very influences which have induced its development? Let the owners of thorough-bred stock, who periodically suffer serious losses, reflect that, no matter how skilful the treatment of such animals might be, it is futile so long as the operation of causes is not taken into account.

In large horse establishments scarcely a fresh animal arrives which is not shortly laid prostrate, not so much by change of locality and food, as by the direct influence of a contaminated atmosphere. After a time, however, even the horse may become habituated to some extent, and present all the signs of health; but if at any time an epizootic should prevail, an animal so circumstanced is always the first and the most seriously to suffer.

With regard to the drainage of stables, I am of opinion that within the buildings they should be on the surface, and that the grating leading to a necessary underground drain should be some feet upon the outside of the stable walls. Where drainage is bad, disease is apt to assume a pyæmic character, and an ordinary inflammation may lead to the formation of multiple abscesses, purulent infiltrations, or even gangrene.

*Work—over-exertion*—by its debilitating effects, tends to induce disease: for example, we have congestion and apoplexy of the lungs, as well as laminitis, myositis, and spasm of the diaphragm, from severe exertion, especially when a horse is not in condition; and from the more continued over-work, as seen in cart-horses, stiffness of the joints and back, unthriftiness, a tendency to and actual development of diabetes, and aortic or heart affections.

Exercise is beneficial to all animals, but when in degree or continuance it exceeds what the strength can bear, or rest can recruit, the animal functions are exhausted and lose their balance, muscular tone is impaired, nervous excitability takes the place of strength, the circulation fails, congestions ensue, the blood is not properly purified, and the various organs are on the brink of disease. Hence it is that animals which do



not actually become diseased from over-exertion are more prone to succumb to the influence of any epizootic that might prevail at the time.

*Want of exercise* is a frequent cause of disease; the muscular system, and with it the circulation of the blood, is the first to suffer, the movements become sluggish, sweats break out upon the least exertion, there is want of condition, in horse phrase, swelled legs, grease; the respiration being but little exercised, the task of decarbonizing the blood falls upon the liver, hence the accumulation of fat and the occurrence of derangements of that organ.

*Heat and cold* are very prevalent causes, and act as follows:—Heat relaxes the whole system; under its influence the muscles, and with them the heart and arteries, lose power and tone, perspiration becomes profuse, the internal organs especially are too much stimulated by blood which has lost more than usual of its water, and less of its hydrocarbon. You will often find that upon the sudden accession of hot weather horses suffer greatly from congestive diseases. In May and June 1867 this was the case in Bradford. The weather had been cold, but very suddenly it became hot, and immediately numerous cases of congestive hepatitis occurred; in other seasons we had to contend with congestive pneumonia. Cold, again, acts as a sedative and debilitant if long continued or severe; it weakens the circulation, especially that of the surface of the body, causes internal congestions, and directly lowers all the vital energies; and the most fatal cases of pleurisy and pneumonia are observed to prevail during and towards the close of severe winters. More specific congestion of the liver is very noticeable in the tropics from this cause. The immense change in temperature between that of the night and the day is very trying in this respect, and the liver, being the largest and most vascular gland in the body, is the one most readily affected. A temporary application of cold to the healthy animal is followed by a favourable and invigorating reaction, but when long continued its effects are most injurious.

## CHAPTER III.

### PATHOLOGY—*continued.*

#### THE MORBID PHENOMENA—SYMPTOMS AND SIGNS OF DISEASE—SEMIOLOGY.

“For my own part” (says Watson), “if I were called to define a symptom, I should say, ‘Every thing or circumstance happening in the body of a sick person, and capable of being perceived by himself or others, which can be made to assist our judgment concerning the seat or nature of his disease, its probable course and termination, or its proper treatment.’ Every such thing or circumstance is a symptom.”

Symptoms are signs of disease, but it is only by mental effort and experience that the practitioner is able to convert symptoms into signs. The idea associated with *symptom* is much more vague than that which is connected with *sign*. Some writers have endeavoured to restrict the word symptom to the phenomena depending on vital properties, whilst those phenomena of disease which are more directly physical are by them called *signs*. Again, some restrict the term symptom to the phenomena manifested by present disease only; but this is contrary to the custom by which we speak of precursory and consecutive symptoms. Symptoms of disease are obvious to all persons alike: for example, the manifestation of pain, the symptoms in an animal suffering from enteritis, are plainly seen by all the surrounding attendants, but it is only the skilled and experienced veterinarian who can detect the expression, the condition of the pulse, &c., the signs by which this malady is diagnosed or distinguished from a mere colic. “Symptoms may be considered as resembling so many *words*. When taken separately, or when put together at random, the words have no force or signifi-

cation. Arrange them in due order, reduce them into a sentence, and they convey a meaning. The sentence is a sign or expression of something which is thus revealed. Symptoms become signs when their import can be interpreted.”—(Sir THOS. WATSON.)

According to the arrangement of Reynolds, symptoms are to be considered as parts of a disease, for he says—“ So long as disease was regarded as some material put into, added to, or engrafted upon the body, the words symptom or sign described the means by which we might recognise the presence of such a material ; but when we define disease as being the sum of changes in function and structure presented by the living being, the words symptom and sign describe only those parts of the disease which are appreciable to the observer’s senses. We call a disease by a particular name, which may express its primary or most important fact, but we cannot separate this one fact from others, as exhibited by the symptoms, but must regard them as integral parts of the malady. Thus the different phenomena of pneumonia, the cough, lung-sounds, respirations, &c., are as much parts of the disease as are the structural changes in the lung. We cannot imagine the existence of symptoms without disease, and *vice versa*.” I must, however, differ from the above-named writer, as I am strongly convinced that when symptoms are thus arranged they are apt to be therapeutically looked upon with too much significance, and thus induce the practitioner to pursue a course of treatment more calculated to modify them than to remove their causes.

Symptoms are *local* or *general*, according to whether they are confined to the diseased part, or affect, more or less, the whole system ; *idiopathic*, when directly proceeding from the disease ; *sympathetic* or *secondary*, when arising from those produced by the primary disease, or from secondary disorder ; *premonitory* or *precursory*, when they precede the full development of disease, generally resulting from the first operation of its cause, such as signs precursory to an inflammation ; *commemorative*, when developed in the previous history of the disease. Again, symptoms and signs are further divided into *diagnostic*, *prognostic*, and *therapeutic*, when they are specially applicable to the distinction, the determination of the event, and the suggestion of the treatment of the disease ; into *objective*, when they present themselves to the scrutiny of the practitioner ; and these

are again divided into the *active* or *dynamical*, when they are only discoverable by motion or manipulation, and the *passive* or *statical*, when they are obvious without such action; *positive*, when they consist of phenomena actually present, and *negative*, when they consist in the absence of phenomena. Diagnostic symptoms are those symptoms which point out the distinction of one disease from another, and the art of *diagnosis* is that by which the practitioner is enabled to arrange *symptoms* into *signs* of disease. A symptom or a set of symptoms which are peculiar to any particular disease, are said to furnish *pathognomonic* signs of such disease, and are called *pathognomonic* or *pathognostic*. A simple symptom is rarely pathognomonic, but two or three conjointly often are so. For example, a discharge is seen to issue from a horse's nose. This is a symptom. It may, however, be due to a catarrhal inflammation, to disease of the teeth, or of the bones of the face; to an affection of the lungs, to a collection of pus in the guttural pouches or facial sinuses, or to glanders. The discharge is so far a *sign*, that it indicates the formation of pus. If we discover, on examination, that the discharge is associated with ulceration of the Schneiderian membrane, with enlargement of the submaxillary lymphatic glands, and more especially if the discharge itself is of a glutinous consistence and starchy appearance, we conclude that it is due to glanders. Taken collectively, these symptoms constitute a diagnostic sign of glanders, and are said to form the *pathognomonic symptoms*.

The interpretation of symptoms requires the closest observation, and in many instances repeated examinations of the patient; for the veterinarian is never assisted by what are termed in human medical practice the *subjective signs* of disease, namely, the sensations felt and described by the patient himself. In veterinary practice all signs and symptoms are objective, and each sign or group of signs has to be duly considered, compared one with another, and each with all; so that a close observation is essentially necessary before a due and proper conclusion can be arrived at and an opinion pronounced. It is by comparing at intervals the various modifications and alterations which occur in the signs and symptoms of disease, that we are enabled to *prognosticate* the nature of its termination, or, in other words, to make our *prognosis*.

## CHAPTER IV.

### PATHOLOGY—*continued*.

#### GENERAL SYMPTOMS OF DISEASE.

##### THE VISIBLE MUCOUS MEMBRANES.

THE general appearance of the visible mucous membranes is of great assistance to the veterinarian in the diagnosis of disease. The natural colour of the Schneiderian membrane and conjunctiva is a palish red, or carnation; any appreciable deviation from this is indicative of some disorder. Increased vascularity and heightened colour—*floridity*—are indicative generally of disturbance and over-excitement of the circulatory system. It does not follow of necessity that disease is present, as the same condition of the membranes is brought about by severe exercise; in fact, any cause of excitement may produce increased redness and vascularity of the visible mucous membranes. This condition is not usually apparent in the tongue and mouth, for the epithelium, more particularly on the tongue, is sufficiently thick to obscure an increased vascularity or stasis.

The appearance of the mouth, however, is occasionally of great importance, *redness* of it being indicative of an irritable and congested condition of the digestive organs generally; vesication, of sporadic or epizootic eczema; a peculiar yellowish or salmon colour, with desquamation of its epithelium, more particularly in the neighbourhood of the incisor teeth and within the lips, of rinderpest; whilst sudden pallor of the mouth and tongue, with coldness, is symptomatic of approaching death from hæmorrhage. The appearance of petechial spots on the visible mucous membrane denotes a depraved condition of the blood, as in purpura. Yellowness of these membranes indicates disease of the liver; *lividity*, a carbonized or non-oxygenated condition of the

blood, as in bronchitis and pulmonary congestion; a *slate-coloured appearance*, a condition of the blood due to the poison of glanders, or a state of the system predisposing to that disease; *rusty colour*, some forms of epizootic diseases; *pink*, or pink-eye, epizootic cellulitis; *pallidity*, anæmia and general debility, or, if occurring suddenly, hæmorrhage. Mere fulness of the capillaries of these membranes, with increased redness, is not always dependent on an increased circulation, for in many diseases, where the powers of the circulatory organs are much diminished, the colour and vascularity may be greatly augmented. In several cases, where death was approaching from a degenerate condition of the structure of the heart, I have noticed that the visible membranes became very greatly congested, and their colour increased; but the shading of the colour has been different to that seen in inflammatory diseases; the circulation in the vessels has been sluggish; the power of the heart—*vis à tergo*—has been insufficient to propel the blood through the minute capillaries; hence the congestion. The purplish line mentioned by some writers as being indicative, when not dependent upon an altered condition of the blood, of debility, has not been present in these cases; indeed the difference, except in the shading of the colour, from the appearance in many inflammatory diseases has been very slight; whilst the state of the heart itself has been recognised by violent palpitations, great irregularity of its action, and almost imperceptibility of the pulse at the jaw and arm. A foul appearance of the mouth and tongue, so valuable an aid to diagnosis in human medical practice, is rarely observable in the horse and ox, but is commonly seen in the dog. However, in some forms of dyspepsia, a slightly foul and soapy condition of the buccal membrane is seen even in the horse and ox, with an acid condition of the salivary secretions, which smell sour and even foetid. Dryness of the mouth is often indicative of inflammatory diseases, more particularly those affecting the organs of digestion. A moist state of the mouth, from excessive secretion of saliva, is symptomatic of disease of the teeth, of catarrhal disease, tetanus, or of the presence of a foreign body, such as a pin or thorn, in some of the oral structures.

SYMPTOMS FURNISHED BY THE CONDITION OF THE SURFACE OF  
THE BODY AND EXTREMITIES.

In a state of health, if the animal is dry and not exposed to extremes of temperature, the surface of the body and the extremities present a warm genial sensation to the touch of the observer, but the variations of temperature in disease are great and important. In severe inflammatory diseases, the legs, ears, and general surface of the body may be cold; the legs and ears presenting a sensation, when handled, of being what is termed "deathly cold." This symptom, if continuous, indicates grave diseases or important lesions; and if, in addition to the coldness of the legs and ears, there be perspiration over the body, or what is termed a cold sweat, the life of the animal is in great jeopardy.

In milder diseases, and those which run through their course slowly, such as many epizootics, peculiar alternations in the temperature of the surface and extremities are generally witnessed: thus one leg may be cold while the other three are warm, or three legs may be cold and one warm, one ear cold and the other warm, &c. &c. This eccentricity is often increased by the warm and the cold condition becoming more or less suddenly changed in situation; the leg which was warm in the morning may be found cold in the evening, the cold leg or legs being warm.

The signs furnished by these alternations are indicative of a depressed and erratic nervous force, and of a tendency to *metastatic* congestions.

A *dry, scurfy appearance of the skin* is often a symptom of indigestion, and of the presence of oxalic acid salts in the blood; and what is called "*hide-bound*," a condition where the skin seems to have lost its pliability and softness, is a symptom of a general state of mal-nutrition, arising from indigestion, improper food, worms in the intestinal canal, or a want of proper exercise, &c. A staring coat often accompanies the above described conditions,—very often a staring coat is the only symptom of ill health. Whenever an animal is seen disposed to shiver, with staring of the coat, upon exposure to moderate cold, or where this tendency exists in an animal whilst not so exposed, in all

probability such an one is bordering on disease. A persistently staring coat, without any other symptom of disease, often indicates the incubative stage of glanders or farcy, and recurrent rigors are generally the fore-runners of strangles or other suppurative diseases. The premonitory stage of lymphangitis is marked by a fit of rigor or shivering, and in one case which came under my notice, acute glanders was ushered in by a continuous shivering fit of three days' duration.

#### THE PULSE.

The pulse is the beating of the arteries, and is usually felt at the jaw—the submaxillary artery, or on the inner side of the arm—the brachial artery. In the cow, whilst recumbent, the pulse may be very distinctly felt on the posterior part of the fore fetlock, and in the dog it is best felt at the femoral, on the inner surface of the thigh.

The pulsations felt by the finger are principally due to the fact that the artery expands during the contraction of the heart, and returns to its previous condition during the relaxation of that organ.

The arterial movements, as indicated by the sphygmograph, constituting the pulse, are described as follows by Dr. Burdon Sanderson:—

“1. At the moment that the heart begins to contract, a vibratory movement of the blood contained in the aorta is produced, in which the molecules of liquid are projected forwards in the axis of the vessel. A similar vibratory movement occurs the moment the ventricles cease to contract. These two movements differ only in this respect, that in the former the primary shock is directed towards the periphery, in the latter towards the heart.

“2. Each of these movements is propagated in the direction of the stream at a rate of about ninety feet per minute,—the one expressing itself at the radial artery by a sudden expansion of the arterial tube, the other by a sudden collapse.

“3. The exact moment at which each movement occurs is indicated by the sphygmograph,—the former by a sudden vertical ascent of the lever, the latter by a descent. In either case the primary movement is followed by a succession of smaller move-



ments in alternately opposite directions. By measuring the time which intervenes between the first and second vibrations, the exact duration of the systole of the heart may be ascertained, for the one occurs at the instant that the ventricle hardens in contraction, the other at the instant that the aortic valve becomes tense in closing.

"4. Both movements manifest themselves with much greater distinctness in some cases than in others. In this difference it seems probable that the respective valves have much to do, and that the systolic vibration is produced by the sudden tightening of the mitral valve, just as the diastolic vibration is due to the sudden tightening of the aortic valve. The precise physical conditions on which the degree of vibration depends, have not as yet been ascertained, but observation points to the conclusion that the intensity of the systolic vibration is greatest when the arterial pressure is lowest at the close of the diastolic period.

"5. From the moment that the ventricles begin to contract, the fulness of the arterial system, and consequently the arterial pressure, rapidly increases. As, however, the arteries at first yield readily to the tide of blood, the tension does not attain its maximum until some time after the hardening of the ventricles in contraction. The duration of the interval between the one event and the other—that is, between the closure of the mitral valve and the moment of highest pressure in the radial artery—varies. It is longest when the arterial system is full; shortest when it is comparatively empty. Hence the measurement of this interval comes to be of considerable importance.

"6. From the moment that the artery attains its greatest distension, it begins to collapse; the form of that part of the sphygmographic tracing which corresponds to the period of relaxation is, as has been recently pointed out by Dr. Rivers, parabolic. In the normal pulse the parabolic form is not easily distinguished, the line of descent being nearly rectilinear; but in all those pulses in which the collapse is rapid, it is very obvious, and most of all in what has been called the monocrotous—single—form, which corresponds to the thready pulse of authors.

"7. In certain conditions of the circulation, the radial artery, immediately after the distension produced by the contraction of

the left ventricle, suddenly collapses, and then as suddenly expands again, the second expansion being sometimes nearly equal in intensity to the first. This constitutes dirotism."<sup>1</sup>—(Dr. BURDON SANDERSON'S *Handbook of the Sphygmograph*, to which the reader is referred for further information.)

I have already stated that the pulse is the beating of the arteries. In each pulsation as felt by the hand the arteries are slightly expanded, distended, and elongated by the wave of blood; sometimes they are laterally displaced, and then return to their original position, after which there is a short period of rest—the interval.

It seems probable that when the heart contracts of itself, it does so gradually and peristaltically, its constituent fibres being brought successively into action; and that, in so far as the movement is deprived of its automatic character by the influence of stimuli acting through the spinal cord, it becomes sudden and instantaneous.—(See on this point Dr. BURDON SANDERSON, or Dr. BELL PETTIGREW'S Paper on the Physiology of the Circulation, in *Lancet*, 1872.) The influences concerned in the production of the pulse are those of the heart, the arteries, and the blood. The heart gives the impulse by which the expanding wave of blood is carried onward, after which the artery contracts in virtue of the elasticity of the yellow fibrous tissue which enters into the composition of its middle coat, or by its additional muscular force.

In health there is a nearly uniform relation between the frequency of the pulse and of the respiratory movements; the proportions being, as nearly as possible, one respiratory movement to three or four pulsations. Thus the pulse of the healthy horse beats about forty times per minute; the respirations are from twelve to fifteen in the same time. In the cow the same relationship does not exist, even in health, between the pulse and respiratory movements; for during rumination it may be observed that the pulse is seventy to eighty a minute, and the respirations not more than ten. Indeed the pulse of the cow in a state of confinement, in so far as regards the number of its beats, cannot be depended upon in the diagnosis of disease; the states of pregnancy and obesity, the effects of artificial food, and of the activity of the lactiferous glands, as well as the excitement caused by the act of rumination, generally produce such

<sup>1</sup> Double pulse.

an impression upon the nervous system as to cause the action of the heart to be much increased, such increase being entirely consistent with a state of perfect health in an animal so circumstanced.

The relation between the pulse and respiratory movements, with the exception above mentioned, is generally maintained where the pulse and respirations are naturally accelerated, as after exercise; but in disease it generally ceases to exist. In many diseases accompanied by increased frequency of the pulse, the acceleration of the respiratory movements bears no comparison to the increased number of the pulsations, and in many other cases the respiration may be very rapid without a proportionate increase in the number of the pulsations; in some instances the pulse may actually fall below its normal standard, whilst the respirations are increased.

The natural pulse of the horse beats about forty times a minute, that of the cow about from forty to fifty, with the exceptions already mentioned, and the pulse of the dog ranges from eighty to one hundred, depending upon the size and breed of the animal, and the pulse of the sheep from seventy to eighty.

The frequency of the pulsations, and the regularity and irregularity of their succession, depends upon the heart, and the pulse at the jaw and arm, when it can be felt, always corresponds to the contraction (systole) of the ventricles, allowance being made for the very slight interval that must elapse before the wave of blood reaches these parts. To the heart's action must also be referred most of the healthy conditions as well as many of the peculiarities in disease; but the vital condition of the arterial system is not without its influence on the character of the pulse.

The arteries are gifted with a certain amount of vital contractility or tone, given to them by their muscular coat, upon which their healthy firmness depends; without this tone they become relaxed and flaccid, yielding readily to the force of the wave of blood, and but slowly recovering their calibre when the expanding wave has passed onwards. It is evident that, in this condition of the vessels, a portion of the impulse of the heart, which is expended in dilating the arteries, is not restored to the blood by the contraction of their coats; the impulse being thus gradually diminished as the arterial wave proceeds on its course,

the pulse becomes weak, and is not in such instances to be considered a proper indication of the cardiac contraction. The contraction may be strong while the pulse is comparatively feeble. As a rule, however, except in valvular incompetency or muscular degeneration of the heart, there is a general correspondence between the cardiac and arterial pulsations, which renders the one a tolerably correct measure of the other. The *quantity* of blood also has undoubtedly some influence on the character of the pulse. When it is deficient, the pulse is likely to be smaller than natural. This is not an absolute rule, for in animals which may be considered to be in a plethoric condition the pulse is sometimes comparatively small. A modification of the *quality* of the blood affects the pulse, and this is most especially exemplified in cases of blood disease, such as hæmo-albumenuria and purpura hæmorrhagica, where it is frequently found that the pulse is "double," "fluttering," or "trembling."

The varieties of the pulse which are described by authors as being recognisable by the touch are—

- (1.) As regards the number of pulsations within a given time, the pulse is said to be frequent or infrequent,—*pulsus frequens, pulsus rarus*.
- (2.) As regards the time which seems to be occupied by each beat, the pulse is said to be quick or slow,—*pulsus celer, pulsus tardus*.
- (3.) As regards the dilatibility of the artery in length and breadth, the pulse is said to be large or small,—*pulsus magnus, pulsus parvus*.
- (4.) As regards the compressibility of the artery, the pulse is said to be hard or soft,—*pulsus durus, pulsus mollis*.<sup>1</sup>

#### THE SYMPTOMS AFFORDED BY THE PULSE.

*The frequent pulse—pulsus frequens.*—Frequency of the pulse, or its number in a given time, varies slightly within the limits of health in animals of the same breed and similar conformation, and more so in animals of different breeds and temperaments. If the standard in the horse be set at forty beats per minute, it is not at all inconsistent with health to find it differ ten beats in two animals differing but slightly in breed and configuration. Thus, one may have a pulse of thirty-five and

<sup>1</sup> Dr. BURDON SANDERSON: *Handbook of the Sphygmograph*.

the other a pulse of forty-five per minute, and both may be in a state of most perfect health; but a difference beyond this indicates some degree of excitement, or some amount of disease.

A frequent pulse—say a pulse above fifty-five per minute—if the horse is not in an excited state, from exercise or other accountable or natural cause, indicates a fevered condition, but not necessarily an increased vigour of the system; on the contrary, a very frequent pulse is generally associated with extreme prostration and debility. A certain quantity of blood is requisite to the existence of the several parts of the body; and when the heart is too feeble to act forcibly, it is compelled to excessive frequency of contraction to compensate for such want of strength. Excessive evacuations either of blood or of any of the secretions are generally attended with frequency of the pulse.

The old belief that “bleeding lowered the action of the heart and arteries,” is disproved by the fact that all evacuations increase the number of the pulsations. I have repeatedly observed the effects of bleeding upon a healthy animal, and found that when from three to five quarts of blood have been withdrawn, the pulse has increased in frequency ten to twenty beats per minute; when the bleeding has been pushed beyond this, say to the extent of from seven to ten quarts, the pulsations have risen to extreme frequency—to 100, 120, or more per minute. As the pulse increases in frequency it becomes diminished in volume, supporting the conclusion that a pulse is small in proportion to its frequency.

*The quick pulse—pulsus celer.*—This term is made use of to denote that condition of the circulation in which the heart accomplishes its contraction almost instantaneously. The terms quick and slow have been objected to by Sir Thos. Watson, as over-refinements, “simply because it was not certain what was meant by the terms by those writers who used them,” and that it was very doubtful whether the quality of quickness or celerity of each pulsation could be truly appreciated by the finger. But the terms are now revived, for the qualities which they express are made manifest by the use of the sphygmograph, an instrument by which the movements of the artery are transferred to paper, in such a manner as to render it possible to measure their extent and relative duration.—(See Dr. BURDON SANDERSON'S *Handbook of the Sphygmograph.*)

Quickness of the pulse differs from frequency. Quickness refers to the time occupied by each beat of the pulse, whilst frequency has reference to the succession of the pulsations. A frequent pulse is that in which the number of beats is greater than usual in a given time; a quick pulse is that in which each beat occupies less than the usual time, though the whole number may not be much increased. Quickness of the pulse is not so difficult to appreciate by the touch in the larger domestic animals as it seems to be in man, and for the reason that the approach to frequency and quickness is normally much greater in man than in the horse and ox. The contrast, therefore, between abnormal quickness and the natural condition is more manifest in the latter than in the former.

Quickness of the pulse is due to the mode in which the ventricles contract, and always indicates irritation and little strength; the heart makes a quick, short contraction, which differs much from the somewhat prolonged, and more or less forcible, contraction of real energy and excitement. The *jerking* pulse is a modification of the quick pulse, and is characterised by a quick, forcible beat, followed by a sudden abrupt cessation. This pulse is found in cases where the nervous irritability is much increased, or it may be due to some deficiency in the aortic valves.

*The slow pulse—pulsus tardus*—or, as it is termed by Dr. Sanderson, the *long* pulse, is the reverse of the quick pulse, and indicates a prolonged or slow contraction of the cardiac ventricles. In inflammatory fever the pulse was formerly described as *frequens magnus et celer*, and was said to differ from that of plethora, which was described as *magnus et tardus*,—distinctions which are not now held to be competent.

*An infrequent pulse—pulsus rarus*.—This kind of pulse is often associated with slowness, and indicates disease of the brain (compression), a disordered condition of the digestive apparatus, and is often indicative of that condition of the system manifested by excessive urination—*polyuria*. In degenerative disease of the heart the pulse is often more infrequent than the cardiac contractions, because the impulse of the heart is too feeble to be transmitted to the arteries.

*The mutual relation of the pulsations* may refer to their succession, their comparative duration, and their strength. In

all these respects the pulse is often irregular, and the source of the irregularity is to be found in a deranged or debilitated action of the heart. An *intermitting pulse* is that in which a pulsation is occasionally omitted. This omission may be regular, occurring at the end of a certain number of beats, or it may be irregularly intermittent, and along with intermission of its beats there may be irregularity in its tone, volume, &c. For example, several beats may occur in rapid succession; these may be followed by others at long intervals, or some beats may be strong, others weak, some quick, others prolonged; and often several run on successively of one character, to be followed by more or less of another character. Occasionally all these irregularities, or most of them, may occur in the same subject. They are indicative of important functional or nervous disorder, or of organic disease of the heart. An intermitting pulse may often be observed in horses presenting all the appearances of perfect health; indeed, an intermitting pulse is very commonly met with in the cart-horses of this city; and, reasoning from the frequency of indigestion, and the nature of their food, one is forced to the conclusion that this irregularity of the heart's action is due to some disorder of the digestive apparatus. When these animals are attacked by any disease, the irregularity of the pulse frequently disappears for a time, or until the animal is restored to its former condition of apparent health.

The *volume of the pulse* may be greater than usual, in which case it is said to be *large*—*pulsus magnus*, or it may be less than usual, when it is said to be *small*—*pulsus parvus*. Largeness or fulness of the pulse may depend upon general plethora, or a prolonged and forcible contraction of the ventricles; to a certain extent on relaxation of the arterial coats, and on obstruction of the capillaries, without diminished power of the heart. It may be associated with *strength* or with *feebleness* of the pulsations. It is important not to confound a full feeble pulse with a full strong one. The feeble pulse is known by the weakness of the impulse, and by the artery yielding to the pressure of the finger. In pulmonary congestion of a severe character the artery is full, and the pulsations feeble. This has been called the "*oppressed pulse of pneumonia*" by veterinarians, and depends upon excessive congestion of the pulmonary vessels. A pulse of this character is improved in tone by a moderate abstraction of blood.

*The feeble pulse*, if associated with softness, the artery yielding readily to the finger, indicates general or cardiac debility. It is sometimes so weak as to give one the idea that the artery is filled with air, hence it has been called the *gaseous pulse*, and is not unfrequent in an anæmic condition of the body.

*The small pulse—pulsus parvus*—may result from anæmia; from congestion of some important organ, as the lungs; from feeble contraction of the heart; or from great tonicities of the arterial coats.

*The hard pulse—pulsus durus*—hardness of the pulse, is that property by which the artery resists compression, and results from contraction of the muscular coat of the arterial walls. Hardness of the pulse is often associated with smallness—*pulsus durus et parvus*; it is then termed corded, wiry, or thready, and this condition is often met with in the earlier stages of inflammatory disease, particularly during the rigor—in endocarditis, and in dangerous inflammations of serous membranes—and, experimentally, the thready pulse has been produced by division of the pneumo-gastric nerves.

Dr. Sanderson describes this pulse as follows:—"When the pulse is small and hard, as during the rigor at the outset of acute diseases, and in certain dangerous forms of carditis, the expansion of the radial artery is sudden and of short duration; the suddenness of the movement not depending upon the rapidity with which the arteries empty themselves by the capillaries, but on the violence with which the heart itself contracts. In this form of pulse there is no second beat. The explanation is clear: the difference between the arterial and venous pressure is so considerable, and the range of variation in the peripheral arteries so limited, that no perceptible diastolic retardation takes place in the capillaries, and consequently no second expansion."

The hard pulse is seldom associated with largeness; the nearest approach to the hard full pulse—*pulsus magnus et durus*—is the strong full pulse of laminitis. It may, however, depend upon hypertrophy of the left ventricle of the heart, and be present independently of any inflammatory disease. When the pulse is very hard, the lateral displacement of the submaxillary artery is very apparent; it is then called by veterinarians a hard rolling pulse.



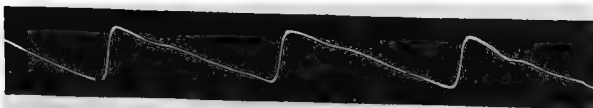


FIG. 1.—Firm and long pulse of health.



FIG. 2.—Hard pulse.

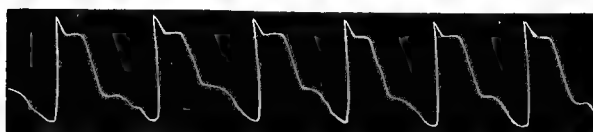


FIG. 3.—Hard and long pulse of hypertrophy of left ventricle.

*The soft pulse.*—Of this kind of pulse several varieties are described. In the small soft pulse, towards the close of progressive exhaustive diseases, the impulse of the heart is slight, the tension of the arteries diminished, and the volume of blood small. In some cases of anæmia, however, or of slow action of the heart, or of great relaxation of the arterial coats, or of these conditions combined, there may be a soft full yet feeble pulse. It is readily known by the feebleness of the impulse, and by the artery yielding readily to pressure.



FIG. 4.—Soft natural pulse.

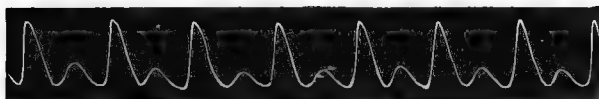


FIG. 5.—Soft frequent pulse of mild fever.

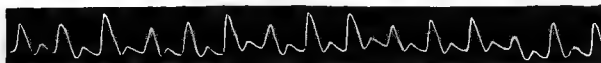


FIG. 6.—Soft frequent pulse of irritative fever.

*The double pulse.*—This pulse is occasionally present in hæmo-albuminuria (red water) in cows, and in purpura in the horse. It denotes, according to Dr. Sanderson, that condition of the circulation in which arterial pressure is diminished, while the venous is increased, and that the capillary current, instead of being constant in its rate of movement, is markedly accelerated during the diastole, and retarded during the diastolic interval. In the undulatory double pulse of typhus, says Dr. Sanderson, the intensity and suddenness of the second expansion appears nearly as great as the first; the explanation being, that in this condition the contractions of the heart are extremely feeble, the arterial pressure being so low that the progressive movement of the blood ceases entirely in the capillaries during the diastolic interval.

*The strength of the pulse* depends chiefly upon the force with which the ventricles contract; in some measure upon the tonicity of the arteries, and upon the volume of the blood. In order to have a strong pulse there must be vigour of impulse, steady resistance to pressure, and a certain degree of fulness. There may be a sharp, forcible beat; but if this is not sustained by a certain amount of subsequent pressure, it indicates irritation rather than energy. A very contracted pulse, however sharp the impulse, cannot be called a strong one. A strong pulse is considered a sign of a sthenic or vigorous state of the system. But it is not invariably so, for hypertrophy of the heart will give strength to the pulse, even though the general powers of the animal be weak.

*Weakness of the pulse.*—In order to have a weak pulse it is essential that the impulse be slight, the tonicity of the arteries diminished, and the volume small. In some cases, however, it will be found, if there be a slow action of the heart, or great relaxation of the arterial coats, that there will be a full, and at the same time a feeble pulse. It is important not to confound this with strength.

*The venous pulse.*—Pulsation of the veins is often seen in the jugulars of the ox, especially during rumination, and seems to be quite compatible with health. If the head or neck of the horse be extended, there will generally be an appearance of a jugular pulse. This, however, arises from the beating of the carotids, which is made visible by the parts being tensely

drawn and pressed together. When a true venous pulse exists independent of heart disease, it is due to the veins filling when the ventricles of the heart contract, and the sudden closing of the auriculo-ventricular valves. It is supposed that there is no actual reflux of blood, but a sudden arrestment of its flow into the right ventricle.

In some forms of heart disease the jugular pulse becomes a diagnostic symptom.—(See *Diseases of the Heart*.)

#### SYMPTOMS CONNECTED WITH THE RESPIRATORY FUNCTIONS.

The respiratory movements may be quickened, difficult or laborious, wheezing, roaring, stertorous, spasmodic or convulsive, irregular, slow, thoracic or abdominal; and be attended with such signs as sneezing, coughing, sighing, or yawning. The breath may be hot, as in pneumonia, fever, &c., and cool or cold, as in cases of great prostration; foetid, as in gangrene and abscesses of the lungs, or in nasal gleet arising from diseased bones or teeth. The matters discharged from the air passages are also signs of great value, as blood, pus, mucus, or a mixture of all of them.

(a.) *Quickened breathing*.—Rapidity of the respiratory movements, although present in many diseases, is easily produced by any cause which accelerates the circulation of the blood, and is generally proportioned to the rapidity of the circulation, and the corresponding need of change in the blood. Thus, exercise, which accelerates the circulation and changes of the blood, also augments the respiratory movements.

(b.) *Difficulty of respiration* (dyspnœa) is a prominent symptom of disease, and may depend upon various causes. In inflammation of the lungs, or pleurisy, there are many circumstances in operation which impede the respiratory movements—as pain, or exudation into the substance of the lungs or cavity of the chest, which mechanically resists the entrance of air into the lungs. Diseases of the larynx or trachea, by diminishing their calibre, are generally associated with extreme dyspnœa, and with sounds, such as roaring or whistling, which furnish pathognomonic signs of the seat of the disease.

Difficulty of breathing, when not accompanied by certain sounds detectable by auscultation, if not sufficiently loud to be

heard at a slight distance from the animal, is not, however, to be regarded as symptomatic of disease of the respiratory organs only, for in many acute diseases the breathing not only becomes quickened, but laboured and difficult to an extreme degree.

Difficult breathing must always be regarded as a symptom of importance, not only as being diagnostic of certain diseases, but as indicating grave alterations in the condition of the blood or in the functions of the nervous system. Diseases are also seriously aggravated, and a tendency to a fatal termination, owing to the non-oxidation of the blood, is augmented by difficult breathing.

(c.) *Stertorous breathing* (Snoring).—This arises from a relaxed state of the velum palati, and is a symptom of brain disease. So long as the respiratory movements are strong and frequent, it does not indicate immediate danger, but when the respiratory powers are impaired, the movements slow—that of inspiration delayed, and then performed with a sudden noise and a jerking effort, and the susceptibility to outward impressions diminished—stertorous breathing is a symptom of the near approach of death. Snoring, so common an accomplishment in the human being during sleep, is but rarely so in the lower animals.

(d.) *Abdominal breathing*, or respiratory movements performed with the ribs fixed as much as possible, owing to pain or mechanical obstruction in the chest, is a symptom of pleurisy, hydro-thorax, and pleurodynia.

(e.) *Thoracic breathing* is that in which the abdominal muscles are prevented from participating to their natural extent in the performance of the respiratory movements. This condition is dependent upon abdominal tumours of great magnitude, ascites, tympanitis, or acute abdominal—peritoneal—pain.

(f.) *Irregular breathing* is that condition where there is a want of harmonious correspondence between the inspiratory and expiratory movements, and is seen in the disease which is commonly known as “broken wind.” The inspiratory movement in this affection is performed quickly and with a jerky effort, whilst the expiratory is performed slowly and with a double action, more particularly of the abdominal muscles. Irregular breathing often becomes spasmodic or convulsive during the paroxysms of the disease.—(See *Broken Wind*.)

Of the phenomena and sounds which attend the respiratory

functions besides those already mentioned, the act of coughing and the varieties of cough demand our careful attention.

A cough is a violent spasmodic action, and the sound is produced by a sudden and forcible expiration, preceded by a firm closure of the glottis; and is for the most part an involuntary effort to remove some source of irritation from the lungs, trachea, or larynx.

The varieties of cough which are met with in veterinary practice are denominated moist, dry, short, hacking, violent, spasmodic, the broken-winded cough, and the cough of the roarer.

*The moist cough* attends cattarrh, bronchitis, and other diseases where the secretion of the mucous follicles of the air passages is increased.

*The dry cough* indicates a non-secretory condition of the air passages, and is usually present during the early stages of catarrhal affections. In pleurisy the dry cough remains throughout the disease; it is also painful, short, and hacking, from the pain which the forcible expiratory movement causes the animal to feel. The dry cough has several modifications from that of broken wind, where it is a miserable apology for a cough, to the loud paroxysmal cough, caused by the irritation of teething, and which may be properly named the dental cough, and the hollow, sepulchral cough of the roarer. Cough does not always arise from direct irritation of the air passages, lungs, or pleura, for we often find an animal coughs when suffering from indigestion, worms in the intestinal canal, diseases of the liver, &c. These sympathetic coughs are always characterised by dryness.

#### THE SYMPTOMS AFFORDED BY THE SECRETIONS.

The secretions may be either diminished, increased, or perverted. In the early stages of inflammatory disease of a secretory organ, its secretory function becomes diminished. Thus, in the first stages of pleurisy the surfaces of the serous membrane are dry and rub against each other, producing the "friction sound;" but as the disease advances some of the natural fluid secretion of the membrane, along with a quantity of inflammatory products in a fluid state, is thrown out, and the surface, which was dry, becomes unnaturally moist. The same changes occur when a mucous membrane or a gland is inflamed. In the febrile state

there is occasionally a diminution of all the secretions, but this can only continue for a short time, as some particular organ, the bowels, kidneys, or skin, is excited to increased action by the presence of effete materials in the blood, and its secretion thus increased, while those of the rest are diminished. General increase of the secretions is scarcely possible, and much more frequently the increase, like the diminution, is confined to one or more secretory organs. Excessive and exhaustive secretions are termed *colliquative*.

*Perversion of the secretory functions* is characterised by an alteration in the character of the secreted materials. The products of secretion are sometimes wholly changed in character from those of the same organ in health, becoming excessively irritant, and yielding evidence to chemical re-agents of the presence of new constituents. In some instances, again, one secretory organ will perform the function of another, as when biliary matters are excreted by the kidneys when the liver ceases to act.

#### SYMPTOMS BELONGING TO THE CALORIFIC FUNCTIONS.

The animal body is so constituted as to be able to bear great extremes of heat and cold, and be yet in itself never greatly above or below the mean standard of health—never greatly above, and never much below, until vitality has ceased, or has been so far reduced as to have surrendered the body to the operation of surrounding influences.

The mean temperature of the body in those internal parts which are most easily accessible—as the mouth and rectum—may be estimated at from 99° to 102° F. In thirty-four healthy horses, all under the same conditions, the temperature was found to be under 101° in ten; 102·2° in one; and 101·2° to 101·8° in twenty-one. In young animals the temperature is commonly about 102°, but in very old ones I have seen it as low as 96°. The external parts of the body become lowered in temperature the farther they are removed from the centre, and are liable to much variation from the state of the surrounding atmosphere.

The method of ascertaining the temperature of the body in the lower animals is by introducing a properly registered thermometer into the rectum. The practice of thus measuring the changes of

the temperature of the animal body has now made progress in veterinary medicine, and is considered of great importance as an aid to diagnosis and prognosis. It has been advocated by clinical teachers since 1754, when Antonius de Haen, of the Hospital of Vienna, impressed his pupils with the necessity of attending to the temperature of the body in disease, *as measured by a thermometer*, instead of being judged by the hand. He showed that even in the cold stage of ague, with the teeth chattering and the body shivering, the temperature of the blood is rapidly rising, although the pallid skin may be cooler than usual, its supply of blood being diminished by the contraction of its vessels.

The production of animal heat is due to certain chemical and vital changes, which are continually taking place in the body; and consist in the absorption of oxygen by the pulmonary capillaries, the combination of that oxygen with the carbon and hydrogen derived from the disintegration of animal tissues, and from certain elements of the food which have not been converted into tissue. This combination of oxygen, or oxidation, not only takes place in the blood, which may be looked upon as a fluid tissue, but in the tissue cells of all parts of the body, the animal heat being thus maintained by the natural changes which are essential to a healthy condition.

The oxidation and natural decay of the tissues, the process by which they are rendered fluid and fitted for absorption, has been termed the secondary digestion, and is thus described by Bennett:—"Growth having been effected, it is necessary that the particles of the tissues which have fulfilled their function and are worn out should be removed to give place to new ones. This constitutes the so-called *secondary digestion*,—that is, in the same way that a piece of food, say flesh, is broken down, rendered molecular and fluid, and is absorbed into the blood to add bulk to the frame, so is our own living flesh constantly breaking down, rendered molecular and fluid, and absorbed into the blood, to be finally thrown out of the system. Thus the blood receives matter from two sources, the primary and secondary digestions; and is continually giving off matter in two directions, one to build up the tissues and form the secretions, the other to produce the excretions."—(BENNETT'S *Clinical Lectures*.)

In many diseases this secondary digestion or metamorphosis of tissues, which is mainly due to their oxidation, is abnormally

increased, and the animal heat unnaturally elevated, and if continued for some time, indicates the existence of the febrile condition. The tissues are rapidly consumed, the blood becomes charged with the effete products resulting from the metamorphosis; the appetite being at the same time impaired, the animal becomes more or less rapidly emaciated, and if the abnormal process be not arrested, naturally or otherwise, finally dies.

The determination of *abnormal* heat in disease has a very great importance, and to detect it practitioners have been in the habit of placing their hand to the skin, and their fingers within the mouth of the patient; but the method of measuring the heat by the thermometer holds a highly important position, not only in cases of illness where the symptoms are apparent, but more particularly in the incubative stages of such diseases as glanders, pleuro-pneumonia-bovina, rinderpest, and other contagious or infectious ailments. Here the early recognition of disease plays an important part in preventing the development and propagation of such maladies, and depends much upon the use of a delicate thermometer. When an outbreak of such diseases as pleuro-pneumonia-bovina or glanders occurs, it is very possible that out of a large herd of cattle or stud of horses but one or two may present any appreciable signs of disease, all the rest remaining apparently healthy; but although they may not present any signs of disease, it does not follow that they are not tainted with the malady in its incubative stage. It must not, however, be supposed that elevation of temperature is diagnostic of any particular disease; it merely indicates the febrile condition, which of itself may be due to a great variety of causes, external and internal, specific and non-specific. Some practitioners are so expert as to diagnose even the incubative stages of pleuro-pneumonia, by placing their fingers in the mouth of the animal, and, from the elevated temperature so felt, be able at once to give an opinion as to the probabilities of the animal being affected, provided always that the disease has already attacked others in the herd. If it be possible to do this by the mere touch, how much more certain and satisfactory will it be, by means of a delicate instrument, to measure the degrees of temperature, and give an opinion based upon conclusions derived from an exact admeasurement.

In glanders and tuberculosis, the two contagious diseases



which the veterinarian is most likely to be asked to pronounce an opinion upon, now that rinderpest is so safely guarded, the examination of all the animals which have been herded or stalled with those labouring under this disease should be systematically made, and all those presenting an elevation of temperature of from two to three degrees should at once be isolated, and looked upon with suspicion, until time proves whether they are suffering from the malady or not. Nothing can be lost by this, but much may be gained, the most important gain being the destruction of the centres of contagion, and the preservation of the remaining stud or herd ; for when an animal is kept mixed with others until the disease be more fully developed, it remains there as a generator of contagious material, and thus propagates the disease over the whole number.

The latest thermometers are capable of registering in half a minute, and the practitioner is warned against using one of the guarded vessels, which it is almost impossible to clean.

Whilst elevation of temperature is generally associated with rapid waste of tissue, it is found that rapid emaciation occurs without a corresponding elevation of temperature, as in polyuria, and that the temperature may be elevated without much increased tissue waste. These phenomena are due to the condition of the "heat centre," which, when active, prevents elevation, but loses its inhibitory power when its condition is depressed.

## CHAPTER V.

### PATHOLOGY—*continued.*

#### ATROPHY AND DEGENERATIONS OF TISSUE.

##### ATROPHY.

ATROPHY implies a wasting of a part, arising from a diminution in the size or a decrease in the number of the tissue elements of which that part is composed. Atrophy of the normal structure of a part may take place without any apparent diminution, but may be coincident even with an increase in size. Thus, the fibres of a muscle may be atrophied, whilst its whole bulk is increased by an interstitial exudation, which, pressing upon the true sarcoous elements, causes their wasting and absorption. Again, a glandular organ may be apparently enlarged, whilst its true gland structures are considerably lessened.

Atrophy is divided into *simple* and *numerical atrophy*. Simple atrophy is applied to the diminution in the *size* of the tissue elements, and numerical atrophy to the process by which their *number* is diminished. The two varieties are often associated. Simple atrophy may, however, exist independently, but the numerical never occurs without the *simple*. Atrophy is not always a morbid process, as, for example, in the disappearance of the thymus gland, which is large in the foetus, and disappears shortly after the birth of the animal.

Simple atrophy is well shown in ordinary emaciation, in which fat disappears from the subcutaneous adipose tissue. Here there is no destruction of the fat cells, but simply an absorption of some of their contents, by which their size becomes more or less diminished. The same process may take place in the cells of glandular organs, and so produce a diminution in the size of

the whole organ. Muscular tissue also wastes in the same way, by a decrease in the size of the primitive fasciculi, as exemplified in the voluntary muscles and the heart.

Numerical atrophy resembles gangrene in some respects, for death of the part is common to both; but they differ in the following points. In gangrene, tracts of tissue are affected. It results from some sudden arrest of nutrition, and a gangrenous mass remains at the termination of the process, in such a condition that the structure can be usually recognised; whereas in *necrobiosis*<sup>1</sup> the change is a molecular one, with gradual exhaustion of the vital power, a molecular disintegration and destruction of elements, so that at the termination of the process all that remains is a glandular *débris*, in which no trace of the natural structure of the part can be discovered. This form of atrophy is of much greater importance than the simple; for in the simple the elements of the part continue to exist, and repair can be effected without any new formation; whilst in the *numerical*, restitution is only possible by the production of new elements.

Inactivity of a part, obstruction of its blood-vessels, failure of its own vital energy, continued pressure upon its surface, and the process of inflammation, are all well-known causes of atrophy. A muscle, if unused, becomes small and pale, and its tissue degenerates. This atrophy of muscular tissue from diminished activity is often seen in the horse. For example, a horse is chronically lame in a foot. In consequence of the pain felt by the animal, the limb is kept in a state of quietude and repose; the animal moves it as little as possible. The muscles, thus kept inactive, diminish in size, and the whole limb seems to shrink. If the cause of the lameness be removed, and the limb regain its former activity, the wasted muscles are restored to their natural condition and strength. Paralysis also leads to shrinking of a limb; and it is recorded that injury to a trunk of the sympathetic nerve will produce the same condition without impairment of motion or sensibility. Atrophy and softening may result from the ingestion of improper food. Majendie kept animals upon food containing no nitrogen and incapable of supplying sufficient nourishment; and one curious result was

<sup>1</sup> The term *necrobiosis* is applied by Virchow to the destruction of histological elements, which occurs both in atrophy and degeneration.

atrophy and softening of the cornea, which melted down and disappeared. Again, the stomach may be softened and its mucous membrane dissolved by the action of the gastric juice. This occurs in the dead body only, although it has been mistaken for the effects of disease. Atrophy may also be caused by the action of medicinal substances, such as mercury, iodine, bromine, and the alkalies. Iodine and mercury are said to exercise an influence on the lymphatic system, and bromine upon the generative organs. Bone is sometimes atrophied both in the horse and in the ox. This atrophy of bone is attended by a diminution in its weight, but the size of the bones in the specimens which I possess does not seem to be altered. The compact tissue is thin, the cancelli have mostly disappeared, the medullary canal, filled with a lardaceous looking fat, extends from one extremity of the bone to the other, and the periosteal surface of the bone presents a more or less cribriform appearance.

The various structures of which an organ is composed may suffer from atrophy. Some, however, do so more than others. The fibrous tissue generally increases in amount. In glands, the secretory cells are the first to suffer. They become small, and often granular, from the presence of oil globules. In muscles, the fasciculi become smaller, and lose their transverse striæ, and finally the whole contents of the sarcolemma may be absorbed, and leave nothing but fibrous tissue, which may or may not be transformed into fat.

#### DEGENERATIONS OF TISSUE.

The wellbeing of every part depends upon the nutritive processes being actively performed by its cells, and upon a due supply of properly constituted plasma—formative material, germinal matter—protoplasm (Huxley), bioplasm (Beale)—being conveyed to it by its vessels. The formative process—nutrition—is performed by the cell, which is the seat of nutrition and function, each individual cell being in itself an independent organism, endowed with all the properties of active life. Various opinions are held as to the constitution of cells. Some writers maintain that a cell is composed of a cell wall, enclosing a cavity containing a nucleus and fluid contents (Schwann, Remak, Virchow, &c.); others state that the existence of a cell

wall is in many cases non-evident, more particularly in embryonic cells, in those of many rapidly growing new formations, blood corpuscles, pus, and mucus; that a cell is a small mass of matter enclosing a nucleus, and that the existence of a cell wall is due to a degenerative process going on in the outer layers of the mass. Thus Beale says, "A cell of epithelium consists of *bioplasm* or bioplasmatic matter, surrounded by *formed non-living* matter, which was once in the *bioplasmic state*. In the same way an oval yeast particle consists of the *bioplasm*, with an envelope of *formed material*, which has resulted from changes occurring when particles upon the surface of the bioplasm died."

Brücke, Stricker, and others consider that the existence of a nucleus is not essential to the constitution of a cell; and they base their conclusions upon the fact that in some of the lowest forms of life cells occur which show no trace of nuclei. Notwithstanding this conclusion—that a cell is an elementary organism capable of exhibiting all the phenomena of life—the nucleus is exceedingly constant in all cells, and presents fewer varieties in form and size than the cells themselves; is spherical or oval in shape, and often contains one or more minute round or angular bodies—the nucleoli; offers greater resistance to chemical reagents than the other constituents of the cell, and in disease often remains after these have been destroyed. It seems to be structurally homogeneous or slightly granular, is more deeply stained by carmine—a property peculiar to formative material—and is in some cases invested by a limiting membrane.

The cell contents have the power of spontaneous movement, and these, as well as the alterations in form characteristic of young cells, are due to the germinal living matter, which is probably the sole seat of the nutritive and formative power of the cell. This germinal matter, or bioplasm, differs in volume and consistence in different cells, and in the same cell at different times. It is capable of imbibing and giving up fluids, and of undergoing corresponding alterations in volume.

It is now generally accepted that every cell originates from a pre-existing cell, that tissue is formed by the natural formation and growth of cells, and that the deviations from this process constitute the basis of every pathological change.

The multiplication of cells may take place—*1st.* By simple division; *2nd.* By budding—gemmation; and *3rd.* By the forma-

tion of new cells within the parent cell—endogenous growth. The multiplication by simple division is the most frequent method. The cell divides and forms two cells, each of which divides again and forms two more, and so on. In nucleated cells the nucleus generally divides first.

In multiplication by budding, a small portion of the germinal matter projects from the cell, and becomes detached by constriction at its base, and forms a new cell.

Endogenous multiplication occurs in cells with an indurated outer layer—dense cell wall—as in some varieties of epithelium. The germinal matter divides within the membrane, and forms a number of new cells. We have thus a parent cell enclosing a brood of young cells, of necessity smaller than the parent cell, which are subsequently liberated by solution of the parent cell wall, or escape by their own inherent power of movement.

Nutrition may then be said to be dependent on two things:—*1st.* Upon a due supply of plasma; and *2d.* Upon the appropriation and elaboration of the plasma by the cellular elements having vital properties, and possessing within themselves the powers of growth and reproduction.

When nutrition becomes absolutely arrested, death of the part where such arrestment occurs is the consequence. When the arrest of nutrition is general, the whole body dies; when it is local, gangrene or necrosis is the result. These two latter conditions having been already described in my work on Veterinary Surgery, I will pass on to those morbid conditions which are dependent on impairment of nutrition, namely, Atrophy and Degenerations.

## CHAPTER VI.

### PATHOLOGY—*continued.*

#### ATROPHY AND DEGENERATIONS OF TISSUE—*continued.*

##### DEGENERATIONS.

ATROPHY and DEGENERATION resemble one another, in so far that, in both, nutrition is impaired and function interfered with. In atrophy the part may be said to be altered in quantity, the waste of the tissue being in excess of the formation of new material. In degeneration, however, the structure of the part is altered in quality, a new formation being present in the tissues, impairing their vitality and interfering with their functional activity.

The degenerations are divided into two classes, namely, the *Metamorphoses* and the *Infiltrations*.

##### THE METAMORPHOSES.

These consist of an alteration in the albuminoid structures, whereby they are changed into new materials, with disappearance of the elements of the tissue proper, softening of the intercellular substance, and loss of function. The metamorphoses are the fatty, calcareous, mucoid, colloid, pigmentary, and amyloid degenerations.

##### FATTY DEGENERATION.

This is the most common of the degenerations, and consists in the transformation of the albuminoid constituents into fat, or rather in the replacement of the healthy tissues of a part by drops or molecules of an oily nature, which appear as

minute granules within the cells of the tissues. A muscular fibre thus affected shows that the sarco- elements, the real

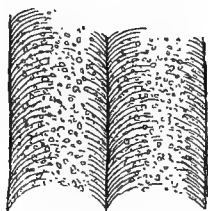


FIG. 7.—Fatty degeneration of muscular tissue. External matter replaced by oil particles.

contractile tissue, within the sarco- lemma, are replaced by shining oil particles, so that the contractile power of such fibre is destroyed. These oil granules, when examined microscopically, present a sharp contour, a dark colour, and strongly refract light. They are soluble in ether; and as they increase, they, by coalescence, sometimes form distinct drops of fat, but usually they retain their granular form. The cells, however, which contain them increase in size and become globular in shape, the nucleus and cell wall are destroyed, and the cell transformed into a mass of fat. The destruction of the cell is an essential element in fatty degeneration, and distinguishes it from *fatty infiltration*, where the cells, within which the fat accumulates, still remain.

This pathological process is very similar to some of the physiological ones. Thus, in the formation of milk, the cells lining the lobuli of the mammary gland multiply abundantly, and are converted into fat; then they break up, and constitute the creamy particles (corpuscles) of the milk. The corpuscles thus formed are pushed forward in the ducts by the formation of new cells, which in their turn undergo the degenerative process, and in this manner a continuous formation and destruction of cells take place.

Fatty degeneration is clearly a kind of atrophy, but is not identical with the simple form, for it may be seen, in degeneration of the heart more particularly, that the muscular fibres may be atrophied with loss of their transverse striæ, and yet contain not a single drop of oil.

When much tissue is affected, the change can be readily distinguished with the naked eye by the yellowish white appearance, and by a diminution in consistence and elasticity.

The parts most commonly affected by the fatty metamorphosis in the horse are the laryngeal muscles—producing the disease which is so well known by the term “roaring,”—the voluntary muscles, particularly those of the shoulder, the coats of the



arteries, the walls of the heart, and the cartilaginous basis of bones.

*Causes of fatty degeneration.*—All influences which interfere with the nutrition of a part may cause this change. Indeed fatty degeneration may be looked upon as an advanced stage of atrophy, and depending upon the same causes—namely, diminished supply of blood, inflammation, decreased functional activity, and diminished nervous influence. Rapid growth is also a cause of degeneration of a part. Thus a rapidly growing tumour is liable to softening and degeneration, as seen in the softening of the central portions of cancer, &c.; and lastly, old age, by impairing the vital activity, may cause degenerations and softening of various parts of the economy.

#### CALCAREOUS DEGENERATION.

*Calcareous degeneration* or *calcification* is an advanced stage of caseation, and is chiefly found to take place in masses of caseous material which are enclosed, and isolated from the influence of the external air, and it is a sure evidence that the growth, whatever may be its nature, has been existent for some length of time. Rokitansky considers the earthy materials not so much as new deposits, but as precipitations of salts, principally consisting of the phosphate of lime and magnesia, and carbonate of lime, from their natural combinations with pre-existent animal matters.

Calcareous degeneration does not always depend upon a previously fatty degenerate state of a part, although it often results from such. It may occur under two opposite conditions, namely, when there is an absolute increase of earthy matters in the blood, and the excess deposited in the tissues; and when there is no such increase, the deposition of earthy salts which then takes place being consequent upon some alteration in the tissue itself. The former condition is oftener met with in man than in the lower animals, and results from extensive caries and other degenerative bone diseases, where the lime salts are removed from the bones into the blood, and deposited in other tissues.

In such cases, the calcification affects many organs simultaneously, and it is not an uncommon thing to find the lungs, kidneys, stomach, intestines, liver, and even the dura mater, infiltrated with lime salts. Again, it is found in man that

chronic disease of the kidneys leads to calcification of several organs. This is due to the fact that the lime salts, which ought to have been excreted by the kidneys, accumulate in the blood, and are deposited in those portions of the tissues which immediately surround the blood-vessels.

Calcification is, however, a process most commonly dependent on local causes—depending not upon any undue quantity of lime salts in the blood, but upon a change in the part itself, whereby the salts which are normally held in solution in the blood are deposited in the tissues. This process is often characterised as an ossification, and not without some reason, as the so-called bone cells or lacunæ are often found in the calcareous substance; they are, however, imperfectly and irregularly developed. I have one specimen in my possession, presented by Mr. Robertson of Kelso, where very well developed lacunæ are found to exist in the calcareous mass. The specimen consists of a bony or calcareous tube surrounding a piece of wood which had been lodged in the inguinal region of a horse for some years. It is a most perfect specimen of the ossification of an exudate, having no connection with a bone.

Calcareous degeneration seems generally to take place in parts whose vitality has been lowered by previous morbid changes, and in degenerating new formations. A part which has become calcified undergoes no further change, but remains as an inert mass: on this account it must be looked upon as a salutary termination of a diseased process. It is thus the most favourable termination to all degenerative diseases, as exemplified in the calcification of many new formations, inflammatory products, tubercle, and atheroma of the arteries.

Rindfleisch states that the cause of the deposition of the salts in the substance of atrophied tissue is partly owing to the stagnation of the nutritive fluids in the part, owing to which the free carbonic acid, which holds the salts in solution, escapes, in consequence of which they are precipitated; and partly to the non-assimilation of these fluids by the degenerated tissue. The saline matters are seen at first as fine molecules, scattered irregularly through the intercellular substance, and are characterised, when viewed by transmitted light, by their opacity, dark colour, and irregularity of outline. They are soluble in dilute mineral acids, after which the original structure of the part, if not destroyed by previous change, may again be recognised.

*Softening* "consists in the liquefaction of the caseous substance, probably owing to some chemical change in its constituents. It commonly occurs in parts which come in contact with the air."—(GREEN.) The caseous mass is converted into a thin puriform fluid containing curd-like matter, composed of animal *débris*, fat, and cholesterine crystals, which, if not absorbed or discharged, may ultimately dry and become calcified.

#### THE MUCOID AND COLLOID DEGENERATIONS

Are conditions which are rarely met with in the lower animals. The reader is therefore referred to works upon human pathology, more particularly to Green's *Pathology and Morbid Anatomy*.

#### PIGMENTARY DEGENERATION.

In this process pigment takes the place of the tissue elements, in the same manner as fat or earthy salts in the fatty and calcareous degeneration.

I have witnessed this form of deposit in the lungs of horses. It is, however, of little importance, and I shall merely refer the reader to authorities upon the subject, amongst whom may be mentioned Aitken, Bennett, Virchow, and Green.

#### AMYLOID DEGENERATION.

This morbid process consists in the transformation of the tissues into a peculiar homogeneous substance, having a relation in some respects to the chemical characters of cellulose or starch.

This degeneration has been long known by medical men under a variety of names, such as the *lardaceous*, *waxy*, *bacon-like*, and albuminoid degeneration. I have met with several cases which presented the physical appearance of this form of disease both in the liver and in the kidneys; but I have only satisfied myself in one instance of the character of the morbid product. For an account of the method by which this disease has been investigated, I take the liberty of making the following quotation from Aitken, who, after mentioning a number of names by which the disease has been known, proceeds to say that "Chemistry and micro-chemical investigation have modified the views regarding the nature of the disease, and now and then have led to modifications in the nomenclature. Under this kind of inquisitive

investigation it has been described—(1.) By Virchow under the name of ‘*animal amyloid*,’ he believing, from the behaviour of the transformed substance with iodine and sulphuric acid, that the substance must be classified with the vegetable carbo-hydrates—cellulose and starch: (2.) Meckel retains the name of ‘*lardaceous*’ or ‘*cholesterine* disease,’ believing that the essential character of the degeneration consists in the development of a peculiar fatty or lardaceous matter of the nature of cholesterine: (3.) The more extended and definite examinations by Friedreich and Kekulé have shown that the substance of the purest amyloid degeneration more closely resembles the *albuminous principles* than any other substance we know of; and (4.) Schmidt has arrived at the same conclusion. The question, therefore, is not yet definitely settled as to the exact nature of the substance into which the tissues are transformed in the so-called *amyloid degeneration*; but the weight of evidence points to its being *albumen* in some form.” The conclusions that the material is allied to albumen are confirmed more recently by Kühne, who, by submitting the amyloid organs to a process of artificial digestion, has completely succeeded in isolating the new material, and has thus been able to determine its albuminous nature. Starch-like bodies (the *corpora amylacea*) have been discovered in various parts of the body, more especially in the nervous system, the ependyma of the ventricles, the white substance of the brain, the choroid plexus, the optic nerve and retina, and the spinal cord of the aged; and larger varieties of the same bodies in the prostate gland of almost every adult, accumulating here sometimes to such an extent as to form large concretions.

The characteristic feature of amyloid is its reaction with iodine, iodine and sulphuric acid, and methylaniline violet. A solution of iodine applied to the amyloid organ causes the affected portions to change to a deep reddish brown colour, which is not, however, permanent, but gradually passes off, and the part regains its former appearance. If the application of iodine is followed by the cautious addition of sulphuric acid, a blackish blue or violet tint is produced. Great nicety of manipulation is, however, necessary to obtain the reaction of the sulphuric acid.

*General characters of tissues which have undergone the amyloid degeneration.*—The cut surface of an organ so affected has a

semi-transparent appearance. It feels like a piece of soft wax, or of lard and wax combined.—(WILKS.) It cuts into portions of the most regular shape, with sharp angles and smooth surfaces, and the thinnest possible slices may be removed by a sharp knife for microscopical examination. They are abnormally translucent. Water and alcohol, acids and alkalies, do not produce any change upon the transformed parts, which may be kept for a length of time without decomposition. The organs affected are increased in volume, in solidity, and in weight, absolute and specific. Anæmia is predominant; but the colour of the blood, or of tissue, shines through the semi-transparent morbid substance.

Amyloid degeneration is generally widely diffused, so much so, that a constitutional state of ill-health seems associated with its production; and in cases preceded by a local disease, such as caries of a bone, the degeneration may be found in the adjacent lymphatic glands only.—(BILLROTH.) This is the earliest appearance of the degeneration yet recognised.—(AITKEN.)

The amyloid substance almost invariably affects the capillaries and small arteries. Their coats become thickened by the deposit, and at last pellucid, transparent, and hyaline. The deposit then extends into the surrounding tissues, and invades both its cells and intercellular substance. The cells become filled with the amyloid material; they gradually increase in size, become round and regular in shape, their nuclei disappear, and they are finally converted into homogeneous bodies, having a translucent or glistening appearance. The effect of this change in the nutrition is to impair or even destroy the function of the affected organ:—*1st.* By obstructing its circulation, by diminishing the calibre of small arteries; and *2nd.* By the pressure of the new material upon its proper substance, whereby secondary atrophic changes are induced. It is important to remember that this form of degeneration is secondary to some serious constitutional disease. It is supposed that it is due to some change in the blood, and that the deposit is in reality an infiltration of an albuminoid or fibrinous substance from the blood into the tissues, which there becomes consolidated. In the human being it is found to succeed chronic suppurative diseases, and the way in which the tissues are affected is as follows:—The change almost invariably commences in the small nutrient blood-vessels, and extends from them to the surrounding parts,

and the general characters of the affection, several organs being simultaneously involved, point to some alteration in the composition of the blood as the cause of the change. In the instance where I succeeded in tracing the nature of the deposit, the horse had been lately bought, suffering at the time from what appeared to the purchaser to be the remains of a cold, sore throat, or strangles. When seen by me, five weeks after purchase, the animal was much emaciated, and presented symptoms of disease of the liver, and was evidently in a dying condition. The *post mortem* revealed the liver much enlarged, weighing over twenty pounds, and of a glistening, palish yellow colour throughout, the new material being stained by the biliary matters; the kidneys were also enlarged, very solid, pale, and translucent in appearance; and when cut, the surfaces were smooth, bloodless, and glistening. The iodine test was most conclusive. It is important to bear in mind that amyloid disease is essentially chronic in its character, and when deposited to any extent, is a sure sign that the animal has been in ill health for a period extending over many weeks, or even months.

#### FATTY INFILTRATION.

This essentially differs from fatty degeneration. In *degeneration*, the fat is derived from a change of the protoplasm itself, leading ultimately to the destruction of the cells of the part. In *infiltration*, the fat is deposited between, as well as within the cells, where it displaces and obscures the nuclei and protoplasm, but does not destroy them. In degeneration, the fat occurs in a granular form within the cell contents. In infiltration, the fat is deposited as distinct drops, which may gradually increase and coalesce, so that the cells, as in the figure, may be completely

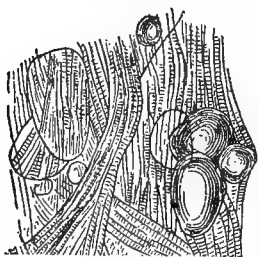


FIG. 8.—Fatty infiltration of heart. Large fat cells intermixed with muscular tissue.

filled and distended with it. The functions of the cells are but little impaired by the accumulation, and are restored to their natural condition by the removal of the fat.

Fatty infiltration is due to an increase of oleaginous materials in the blood, which arises from high feeding and want of exercise. It occurs as a physiological process in the fattening of animals, where the connective tissue becomes filled with fat.

In the human being it has been observed that this form of infiltration is occasionally owing to the presence of fat in the blood, arising from its absorption from some particular tissue and its deposition in another: for example, the general emaciation of pulmonary phthisis is sometimes associated with fatty infiltration of the liver. "Why the deposition should take place in certain tissues, and the fat be removed from some and deposited in others, is not known."—(GREEN.) Fatty infiltration, more particularly of the liver, has occurred in my experience involving legal points of some consequence. In two cases which more particularly occur to me, the animals died within a few days after being purchased. In one case the liver weighed thirty-five pounds, was of an opaque yellowish white colour, its surface smooth, the edges round and thick, doughy to the touch, pitted on pressure, and when cut into, the knife was coated with oil, which oozed from the cut surfaces pretty freely. In the second case, death occurred twelve days after purchase: the liver was not so large as in the former instance, the deposition occurring more as isolated patches than as a general infiltration. In both instances the purchasers succeeded in obtaining a return of the purchase-money. Fatty infiltration is caused by high feeding, more particularly by food containing an excess of the hydrocarbons—such as the oilcakes—and is apt to occur in animals quickly prepared and made fat for sale. It also frequently occurs in very highly fed cart-horses employed for slow work, and often causes death by the liver enlarging to such an extent as to burst its capsule.

Why the liver should suffer from the accumulation of fat, is explained by the fact that when animals are fed on hydrocarbonaceous food, an excess of fat in the blood is the consequence, such fat being, before it is finally disposed of, deposited in the hepatic cells, more particularly in those cells in close contact with the capillaries of the vena porta, at the circumference of the hepatic lobules. This temporary infiltration occurs after every meal, and the fat passes from the cells at the

circumference to those situated more within the liver, then into the blood-vessels, and is finally disposed of, either by being deposited in other tissues, or destroyed in the maintenance of the animal heat.

There is thus a temporary accumulation of fat in the liver, which is gradually removed, and the vitality of the hepatic cells is not impaired. By bearing these facts in mind, we can the more readily understand that when an animal is constantly fed on such a diet, the accumulation may be too great to be disposed of or consumed, and that it remains permanently within the hepatic cells, or so long as the animal continues to be so fed and treated, interfering to some extent with the secretory powers of the organ, and rendering it unfit for ordinary work. A liver subjected to the stimulating influences of such food is prone to attacks of congestion, which are occasionally so great as to cause a rupture of Glisson's capsule, and bring on death from hæmorrhage. It is well to remember that when a horse is excessively fat, the liver may be in this condition; that there is a possibility of death occurring from congestion or rupture; and that the probabilities of recovery from diseases which may have no immediate connection with the liver itself are much lessened. If, however, the animal be carefully brought into condition, regularly but moderately worked or exercised, and fed on food containing no excess of the hydrocarbons, but rich in nitrogenous materials—such as oats, beans, and hay—the liver cells will be again restored to their normal condition by the absorption of the contained fat.

As a veterinary legal question the matter is of some importance, as horses often die if not cautiously and carefully treated when loaded with fat.

It is well known that a practice prevails in many parts of the country of fattening horses rapidly prior to showing them for sale, by giving them an immoderate quantity of highly carbonaceous or even oleaginous food. To the eye, such animals present all the signs of good health. They are, however, totally unfit for work, and too well does the experienced veterinarian know, that when they are seized by an ordinary attack of disease—such as a cold, influenza, &c.—that the chances of recovery are more remote than they otherwise would be, by the liver and other internal organs being loaded with fat. The ten-



dency to produce fat rather than muscle prevails to a very great extent in this country, encouraged and fostered by our agricultural societies, and by purchasers who buy to "please the eye." The question presents itself—Who is responsible when a horse dies in a few days after purchase, either directly or indirectly from this cause? The buyer or the seller?

Fatty infiltration must be carefully separated from fatty degeneration. Degeneration of normal tissues is at all times a morbid process indicative of chronic disease. Infiltration, on the other hand, is a physiological process depending on well-known causes, and furnishes no evidence of a chronically diseased condition of the organ affected. As an example of the importance of the separation of the two conditions, we will suppose a case analogous to what often happens, of a horse in poor condition—thin—being purchased from the breeder and rapidly fed for sale. Such an one puts on fat quickly, and thrives as well as its purchaser can desire. We will say that it is sold and put to work, and that it dies in the course of a week or two after the second sale. Altogether it has not been out of the hands of the breeder more than two months. Now, the last purchaser naturally looks for restitution to the person from whom he bought it, who, in his turn, may make a claim on the breeder. In many cases the matter is amicably settled by the three interested parties dividing the loss amongst them. Is this just? Decidedly not; and in all such instances the man who sold the horse before the fattening process had been commenced should not be made to suffer, the enlargement of the liver being merely due to an accumulation of fat, and not to any disease: and if one individual is to suffer, let the man who resorted to the forcing system of producing fat be made responsible; or let the loss be divided between him and the man who bought what pleased his eye, and who must have known, or ought to have known, that an animal loaded with fat was unfit for useful purposes. But should an examination reveal that the condition of the liver was due to degeneration, the case is entirely different, for beyond a doubt the disease must have been present prior to the first purchase, and the breeder ought then to be responsible for the whole loss.

Degeneration generally occurs in the aged, and may be associated with general emaciation, and with degeneration of other

organs. Infiltration occurs in both the young and old, and is associated with a general obese condition of the whole system.

#### NUTRITION INCREASED.

Opposed to the conditions of atrophy and the degenerations, is that of increased nutritive activity, or *hypertrophy*. In hypertrophy there is an absolute increase of the normal tissues of a part, resulting from an increase in its functional activity.

Hypertrophy, although sometimes depending on disease, is of itself a compensating process, whereby injury from damage to an important organ may be avoided; as, for example, in the thickened condition of the heart in disease of the valves, and in the same condition of that organ when not associated with any disease, but arising from the animal being repeatedly forced to severe trials of strength and endurance, in racing, hunting, &c. In the first instance the hypertrophy of the cardiac walls arises from the necessity of a more powerful contraction for the purpose of overcoming valvular obstruction, and sending a due supply of blood to the various organs of the body, and in the other case of contracting powerfully to attain the same end, during repeated and severe exercise.

As a physiological process, hypertrophy of muscular tissue is produced by the process of training, where the muscles of the whole body are enlarged, strengthened, and made capable of performing extraordinary feats of strength and endurance. Who can fail to see in a well-trained racehorse the muscles standing, as it were, prominently one from another about the quarters and thighs, "hard as iron" to the touch, and giving the animal an appearance of being thin and poor. The appearance of thinness is the result of the great prominence of each individual muscle, of increase in their volume and weight, and an extraordinary healthy tonicity.

"The cause of real hypertrophy seems to be always the increased exertion of the organ; more than usual effort is demanded of it, and according to the law of the circulation, which we have noticed, more blood flows to the part than usual. This, if the organ be in a healthy state, not only supplies its waste, but furnishes material for increase and development. The heart in various diseased states of its valves, the urinary bladder in stric-

ture of the urethra, the remaining kidney when one is destroyed, the muscles, and even the solid bones themselves, when long and actively exercised, afford excellent examples of true hypertrophy."—(C. HANDFIELD JONES.)

Whilst increased nutritive activity may lead to hypertrophy, or an increase of normal tissue only, it may, on the other hand, lead to the production of new materials in a part, more particularly of fibrous tissue, which in its turn may contract upon the tissue of the organ and cause it to atrophy.

## CHAPTER VII.

### PATHOLOGY—*continued.*

#### CHANGES IN THE BLOOD.

**HEALTHY BLOOD**, when it can be seen flowing in the vessels of a living part, appears as a colourless fluid—the *liquor sanguinis*—containing minute solid particles—the blood globules—of which the greater part are red, and the remainder colourless or white.

Analyses of blood show that it is composed of, in 1000 parts—

Water,	.	.	.	.	.	784
Red corpuscles,	.	.	.	.	.	131
Albumen,	.	.	.	.	.	70
Saline matters,	.	.	.	.	.	6.03
Extractive matters,	.	.	.	.	.	6.77
Fibrin,	.	.	.	.	.	2.2
						1000.00

The above constituents being proportioned as follows:—

Water,	.	.	.	.	.	784
Albumen,	.	.	.	.	.	70
Fibrin,	.	.	.	.	.	2.2
Red corpuscles,	{	Globulin,	.	.	.	123.5
		Hæmatin,	.	.	.	7.5
		Cholesterine,	.	0.08		
		Lecithin,	.	0.42		
Fatty matters,	{	Oleic and margaric acids,				1.3
		Volatile and odorous fatty acid,				
		Fat containing phosphorus,				
		Chloride of sodium,	.	.		3.6
		Chloride of potassium,	.	.		0.36
		Tribasic phosphate of soda,	.	.		0.2
Inorganic salts,	{	Carbonate of soda,	.	.		0.84
		Sulphate of soda	.	.		0.28
		Phosphate of lime and magnesia,				0.25
		Oxide and phosphate of iron,				0.5
Extractive matter, urea, glucose, gases, etc.	.					5.47
						1000.00

These several constituents are subject to some variations consistent with health. Thus the water varies in quantity according to the period since food has been eaten, the amount of bodily exercise, the state of the atmosphere, and all other events that affect the ingestion or the excretion of fluids. According to these conditions, it may vary from 700 to 800 parts in 1000. On the whole, however, a degree of uniformity is maintained, because nearly all those things which tend to lower the proportion of water in the blood, such as active exercise, or the addition of saline and other solid matter, excite thirst; while, on the other hand, the addition of an excess of water to the blood is quickly followed by its more copious excretion in sweat and urine.

Dr. Zimmerman has shown that the quantity of water in the blood is increased during its abstraction from an artery or vein. In bleeding dogs he found that the last drawn portion contained 12 or 13 parts more of water in 1000 than that first drawn. This experiment goes to prove that the fluid of the tissues must be rapidly absorbed during the operation.

The water in the economy is required for many important purposes. (1.) The proper viscosity of the blood, and the degree of its adhesion to the blood-vessels, is due to the presence of water in proper proportion. (2.) It determines to a great extent the volume of the blood. (3.) Water is the general solvent of the other materials of the liquor sanguinis. (4.) The activity of absorption by the blood-vessels depends upon the due fluidity of the materials to be absorbed, for it is well known that no fluids quickly penetrate the vessels but such as are of lesser density than the blood.

If the presence of water in excess continues beyond a short period, it is a certain symptom that the other constituents are defective in quantity, and that the condition termed anæmia is present.

Deficiency of water in the blood is witnessed in super-purgation, diarrhœa, &c. The deficiency, however, is generally of a temporary character, for nature prompts the animal to overcome it by drinking freely. The water also becomes deficient when an animal sweats profusely from any cause, and when prevented from allaying his thirst for a considerable period, as when hunting or going long journeys. A great mistake is made

by those in charge of horses who have a belief that it is a bad thing to allow a horse to drink while out on a journey or in the hunting-field. Small and repeated drinks are always beneficial, and prevent much suffering.

The *albumen* of the blood may vary, consistently with health, from 60 to 70 parts in 1000. Its amount, according to Andral and Gavarret, is notably increased in various diseases; but this excess does not appear to be characteristic of any. In acute rheumatism an increase was found varying from 4 to 24; in pneumonia the highest increase was about 12; in pleurisy the extraordinary amount of 34 in excess was once observed; and several other instances are mentioned of a lower degree. Excess of albumen in the blood is witnessed in the horse in a disease which the late Mr. Haycock termed hysteria.—(See *Azoturia* or *Uræmic Paralysis*.) Very probably the albumen in this disease is not only excessive in quantity but deteriorated in quality.

The albumen is defective in cases of piroplasmoses and trypanosomiasis, in the “rot” in sheep, and in dropsies, whether dependent on organic disease of the heart, or on the presence of entozoa—distomata—in the liver. It is a remarkable fact, recorded by Andral, Gavarret, and Delafond, that in sheep affected with anæmia, with deficiency of the red globules, but without dropsy or entozoa, there was no deficiency in the albumen. Dr. Williams concludes from this, that cachexia and anæmia attended by dropsy owe this concomitant to a want of albumen in the blood; and he seems to be of opinion that a deficiency of the albumen, by destroying the due density of the blood, and allowing it to transude through the coats of the vessels, is a chief feature in the dropsical diathesis.

*The fibrin of the blood.*—When fresh-drawn blood is left to itself, it very soon coagulates or becomes solid, and the substance thus becoming spontaneously solid is the fibrin, which, when separated from the blood, is a tough, elastic, tolerably firm, whitish-grey looking material, insoluble in water. Under the microscope the fibrin appears as a fibrillated matter. It is stringy and elastic. The white globules are usually seen imbedded in the mass, but they do not appear to modify the character of healthy fibrin. This coagulation is due to the formation of fibrin, a material which does not exist in the blood circulating in healthy vessels, but is formed in the blood and lymph when removed from the body. At first

the crassamentum or clot is soft, consisting of a few fibres on the surface. These soon extend throughout the mass, which becomes jelly-like. In the horse coagulation takes place but slowly, and the red corpuscles consequently sink; the upper surface, being thus deprived of their colour, assumes a straw-coloured appearance, and is called the buffy coat. Although the whole mass solidifies, the fibrin is only present in quantity of about 3 in 1000.

MM. Andral and Gavarret record that in the domestic animals the fibrin diminishes before but increases after parturition. In the human female, however, it seems that the fibrin is decreased during the first six months of pregnancy—the average being 2·3; whilst in the last three months it is so increased as to average 4. Fibrin is more abundantly formed in arterial than in venous blood; and it is mentioned by Schmidt that the blood of the portal vein contains only one-third of the ordinary amount of fibrin found in the blood of the jugular vein.

*An excess of the fibrin*—hyperplasma or hyperinosis—exists in all true inflammations. In acute rheumatism it is generally very high; in some cases, according to MM. Andral and Gavarret, as high as 12 parts in the 1000. In some exhausting diseases, and even in anæmia, the fibrin is found occasionally increased in quantity. In rapid emaciation, more especially when accompanied by total loss of appetite, the quantity of fibrin is very materially increased. This is explained by the fact that fibrin results from the metamorphosis of the albumen of the blood and of the tissues. An increase of fibrin is found in meagre, half-starved horses, amounting to 7 or 8 above the healthy mean. In one case, where no food was given for four days, it was found increased from 5 to 9. As the fibrin is increased by all inflammatory diseases, such increase can also be artificially induced by the application of external irritants, such as blisters, setons, or the actual or potential cauteries. This fact is of great importance therapeutically, and it should be borne in mind that the application of the so-called counter-irritants in extensive inflammatory diseases, tends to increase a condition of the blood which is already present to an abnormal extent.

Another very important circumstance connected with the increase of fibrin in the blood is pointed out by Andral, namely, that the effect of bleeding was not to diminish it. In M. Andral's experiments the red globules became diminished in number by

each abstraction of blood, whilst the fibrin often rose, sometimes considerably. The following examples are very conclusive:—

	First Bleeding.	Second Bleeding.	Third Bleeding.	Fourth Bleeding.	Fifth Bleeding.	Sixth Bleeding.
Acute Rheumatism,	6·1	7·2	7·8	10·2	9·0	7·0
Pneumonia, .	7·1	8·2	9·0	10·0	—	—
Peritonitis, .	3·8	4·7	6·1	—	—	—
Pleurisy, . .	3·9	5·8	—	—	—	—

MM. Andral and Gavarret found that the fibrin production was increased in tubercular disease. In the crude state of the tubercles, the mean is about 4, when softening has commenced it is about 4·5, and when cavities have formed it is from 5 to 5·5, the red globules decreasing from the first.

*Deficiency of fibrin*—hypoplasma, hypinosis, or defective coagulating power—is observed in various morbid states, in disease, and in conditions bordering upon it, and is indicated by imperfect coagulation of the blood when drawn. It is deficient in all conditions where the blood is imperfectly arterialized; and this is in accordance with the physiological law that arterial blood is more fibrinous than venous. Thus, in death from asphyxia, the blood often remains semi-fluid, of a dark, tarry appearance, and coagulates only after being exposed to the air and absorbing oxygen. Various poisons seem to have the power of preventing the coagulation of the blood. Among these are hydrocyanic acid, carbonic acid, sulphuretted and carburetted hydrogen, and ammonia. Andral states that if a concentrated solution of carbonate of soda be injected into a vein, the animal presents the symptoms of blood disease, and that the blood is found fluid in the vessels. The same result may be produced by arsenious acid, digitalis, and oxalic acid. Other substances, although analogous to the carbonate of soda, seem to have no effect. Thus, the nitrate of potash does not always impede the coagulation of the blood; and Dr. Handfield Jones states that he has observed that liquor potassæ, although given for several days, and until the animal's health was interfered with, did not at all interfere with the coagulation of its blood; and he infers from these observations that it is not the alkalis, as such, nor the neutral salts, as such, which produce the effects usually ascribed to them upon the blood, but certain substances of



particular qualities. In low fevers, influenzas of a typhoid character, particularly when animals so affected are stabled in ill-ventilated houses, the blood becomes remarkably fluid, and gravitates, even in cases that recover, to the depending parts of the body. Thus we find that in the so-called purpura hæmorrhagica the extremities and inferior portions of the abdomen, breast, and face become the seats of extensive swellings, caused by an accumulation of a preternaturally fluid blood in the vessels, and even in the tissues, whilst the appearance of petechiæ, vibices, and the occurrence of hæmorrhages are due to the same cause. The partial suffocation to which animals are sometimes exposed when confined in buildings accidentally or otherwise burnt, produce a remarkable degree of fluidity in the blood. I have often witnessed this in horses and cattle which had been early removed from burning buildings,—in fact, when they had not been exposed to any fire, but to the effects of the smoke of smouldering straw, wood, &c. At the time, the symptoms of distress have not always been noticeable, and little has been thought of the matter. In the course of time, varying from a few hours to three days, the animals have presented symptoms of great respiratory disturbance, with hæmorrhage from the vessels of the lungs, the blood presenting a dark olive appearance, and coagulating but very imperfectly, even when exposed to the influence of the air. In such cases, it is quite fair to presume that the coagulating powers of the blood have been destroyed by the action of the products of combustion contained in the smoke, and that it—the blood—gradually accumulates in the vessels of the lungs until they have become congested, even to rupture of their walls. In apoplexy of the spleen and in “septicæmia” the same hypinotic condition of the blood is seen, and is probably the cause of the congestions and extravasations which characterise those diseases. Although it may justifiably be said that imperfect coagulation, as well as the abnormal fluidity of the blood, depends upon the absence of fibrin, modern research has proved that fibrin, as such, does not exist in the blood, but is formed by the combination of two substances—or two modifications of the same substance—namely, *globulin*<sup>1</sup> and *fibrinogen*; the former of which existing in the serum of the blood, and in some tissues of the body; whilst the latter, so

<sup>1</sup> Fibrino-plastic.—(SCHMIDT.)

far as it is known at present, exists only in the plasma of the blood, of the lymph, and of the chyle, and in fluids derived from them.

“Coagulation of the blood” (says Professor Huxley) “is an altogether physico-chemical process, dependent upon the properties of certain of the constituents of the plasma, apart from the vitality of that fluid. This is proved by the fact that if blood-plasma be prevented from coagulating by cold and greatly diluted, a current of carbonic acid passed through it will throw down a white powdery substance. If the white substance be dissolved in a weak solution of common salt, or in an extremely weak solution of potash or soda, it after a while coagulates, and yields a clot of pure fibrin. It would be absurd to suppose that a substance which has been precipitated from its solution, and re-dissolved, still remains alive.

“There are reasons for believing that this white substance consists of two constituents of very similar composition, which exist separately in living blood, and the union of which is the cause of the act of coagulation. These reasons may be briefly stated thus:—The pericardium and other serous cavities in the body contain a clear fluid, which has exuded from the blood-vessels, and contains the elements of the blood without the blood corpuscles. This fluid sometimes coagulates spontaneously, as the blood-plasma would do, but very often shows no disposition to spontaneous coagulation. When this is the case, it may nevertheless be made to coagulate, and yield a true fibrinous clot, by adding to it a little serum of blood.

“Now, if serum of blood be largely diluted with water, and a current of carbonic acid gas passed through it, a white powdery substance will be thrown down; this, re-dissolved in a dilute saline, or extremely dilute alkaline solution, will, when added to the pericardial fluid, produce even as good a clot as that obtained with the original serum.

“This white substance is called *globulin*. It exists not only in serum, but also in smaller quantities in connective tissue, in the cornea, in the humours of the eye, and in some other fluids of the body.

“It possesses the same general chemical properties as the albuminous substance which enters so largely into the red corpuscles, and hence at present bears the same name. But

when treated with chemical re-agents, even with such as do not produce any appreciable effect on its chemical composition, it very speedily loses its peculiar power of causing serous fluids to coagulate. For instance, this power is destroyed by an excess of alkali, or by the presence of acids.

"Hence, though there is great reason to believe that the *fibrino-plastic globulin* (as it has been called) which exists in serum does really come from the red corpuscles, the globulin, which is obtained in large quantities from these bodies by the use of powerful re-agents, has no coagulating effect at all on pericardial or other serous fluids.

"Though globulin is so susceptible of change when in solution, it may be dried at a low temperature, and kept in the form of powder for many months without losing its coagulating power.

"Thus globulin, added, under proper conditions, to serous effusion, is a coagulator of that effusion, giving rise to the development of fibrin in it."

The latest theories of coagulation slightly modify the above. Thus Schmidt says that the fibrin is formed by the coming together of two proteid substances which occur dissolved in the plasma, viz.—(1) *fibrinogen*, i.e., the substance which yields the chief mass of the fibrin; and (2) *fibrino-plastic substance*, or fibrino-plastin. The latter terms are now rarely used, having been replaced by either of the following—serum-globulin or paraglobulin. In order to determine the coagulation a ferment seems necessary, and this is supplied by (3) the *fibrin ferment*.

Both fibrinogen and fibrino-plastin belong to the group of proteids called globulins. They are insoluble in pure water, but are soluble in dilute saline solutions (common salt, 1 per cent.), phosphates of the alkaline earths, and calcium sulphate. They are not distinguished from each other by any marked chemical characters, but differ in the following particulars: Fibrino-plastin or paraglobulin—serum-globulin—is only appreciably precipitated when a solution of sodium salt of at least 20 per cent. has been added, whereas fibrinogen is easily precipitated by the solution of salt (sodium chloride), when it only reaches 16 per cent.

Again, the very light granular precipitate of paraglobulin is more easily dissolved than that of fibrinogen, and its solution only begins to coagulate at 133° F., whereas that of fibrinogen

readily coagulates at 105° F. The precipitate of paraglobulin forms a granular powder, whilst that of fibrinogen adheres to the sides of the vessel as a sticky deposit.

The theory of Schmidt now seems to be superseded by that of Hammersten, and his researches have led him to conclude that fibrino-plastin is quite unnecessary for coagulation, and that the fibrin is formed from one body,—viz., fibrinogen, which is present in plasma when it has been acted upon by the fibrin ferment, which, however, has not as yet been obtained in the pure state.

Neither Hammersten nor Schmidt assert that this body is of the nature of a ferment, but they use the term for convenience. It is quite certain that fibrin may be formed, when no fibrino-plastin is present, by the addition of calcic sulphate or casein prepared in a certain way; but as yet the views of specialists on this subject are by no means settled. Halliburton argues that the ferment supposed to be the determining agent in coagulation is really itself a proteid—in fact cell globulin; but as several experiments of others seem to show that the salt extract of fibrin can be very completely freed from all proteid matter without any loss of fermentive activity, this ferment can scarcely be a globulin. On the other hand, it might be assumed that the coagulative power is really due to the calcium sulphate (GREEN), which salt is well known to favour clotting even in very dilute solutions. But finally, as against this latter supposition, remains the fact that in solutions of the fibrin ferment which have been most carefully freed from everything giving proteid reactions, and also carefully freed from saline matters by dialysis, the power of inducing coagulation only becomes lost after heating to about 154° F., a condition that could by no means influence calcium sulphate, even if traces of it were present. Buchanan in 1831, Mantegazza in 1871, and Schmidt a year later, had all suspected that the white cells were the source of a ferment connected with coagulation.

Among several recent methods of separating the ferment, perhaps the best is the prolonged action of strong alcohol—fifteen times the volume of the serum used—for two weeks or more. Subsequent filtering, drying and powdering the precipitate, and dissolving it in distilled water to twice the original volume of the serum, gives an extract with which coagulating experiments can be made.

In addition to those above named, Halliburton and Rauschenbach consider that plasma entirely free from white cells cannot be made to coagulate in the usual way, but information on the fibrin ferment is much less accurately determined than is the case with almost any other known inorganic enzyme.

#### FERMENT CONTAINED IN THE SECRETIONS OF ANIMALS AND PLANTS.

The following appears to be all that is really known:—(1.) Temperature is of much account, action stops after  $132\frac{2}{3}^{\circ}$  F. has been reached: 2. The amount of coagulative change is not affected by the quantity of the ferment, only the rate, as with all known ferments; when the quantity is small the clotting is slow: (3.) The ferment is precipitated along with gelatinous matter found in its solutions; 4. It is most probably not used up, and a very small quantity indeed seems to be sufficient.

It seems, however, to be the general opinion that the ferment is perhaps derived from the disintegration of the white corpuscles when blood is shed, or within the economy in certain conditions, such as septic fevers, diseases of the blood-vessels, foreign bodies introduced into them, pleuro-pneumonia contagiosa, the microbe of which seeming to determine coagulation of the blood within the vessels, and of the exudate in the surrounding structures of the lungs, *i.e.*, parenchyma cells, alveoli, and bronchi; and it is also found that an injection of ichor into the blood causes the destruction of an enormous number of corpuscles, resulting in spontaneous coagulation within the circulation, after which the coagulability of the blood is diminished. From the foregoing we can understand how in metritis and other septic diseases where thrombi are abundantly formed, the blood itself may coagulate more feebly than natural.

Whilst fibrin is absent in healthy blood, it must be admitted that the materials from which its factors are derived must be contained in their proper conditions and proportions. But it is equally evident that circulating blood contains no ferment so long as both the fluid and the walls of the vessels are in a normal condition.

Coagulation of the blood varies in different animals, and is

modified by disease. The blood of the horse, for example, coagulates more slowly than the blood of other animals and of man; hence what is termed the buffy coat is observable in coagulated healthy horse's blood, the buffy appearance on the top of the clot being ascribable generally to the fact that slow coagulation gives time to the blood globules, which are of higher specific gravity than the liquor sanguinis, to sink towards the bottom of the vessel before being incarcerated in the meshes of the coagulating fibrin. To prove this, fill two vials with the blood of a healthy horse, and to the blood in one add a small quantity of the chlorate of potash, and leave the other to coagulate naturally. In the course of a very short time the blood to which the chlorate has been added will be found firmly coagulated, and with but a slight or without any buffy coat; whilst the blood to which no addition has been made remains fluid for a much longer time, the globules falling to the lower parts, leaving the upper portion of the clot a straw colour.

During the first stage of coagulation, which is termed that of gelatinisation, the clot appears as a semi-solid mass. In the course of some time, however, it divides into serum and clot (*crassamentum*), the serum being pressed out by the fibrin. This shows that the fibrin possesses the power of contraction; and to the presence or absence of this power the further changes in the clot are due. The clot may be defective in solidity—gelatinous—or it may present an excess not only of fibrin, but of contracting properties. The shrunk, contracted, or cupped appearance of the clot indicates increased contractility and a sthenic state of the system generally; whereas the gelatinous clot is indicative of an aplastic condition of the blood and an asthenic state of the system.

## CHAPTER VIII.

### PATHOLOGY—*continued.*

#### CHANGES IN THE BLOOD—*continued.*

##### ANÆMIA, HYDRÆMIA, OLIGÆMIA.

THESE terms are applied to that condition of the system in which there is a deficiency and poverty of the blood, a state brought about by loss of blood (hæmorrhage), profuse discharges of any of the natural fluids of the body, as those of diabetes, in which it seems as if the blood globules were melted down to supply the profuse discharge; insufficient food, deprivation of fresh air, exhausting diseases, and the action of some deleterious agents, as witnessed in horses kept in ill-ventilated stables, or suffering from the influence of the glanders poison.

The symptoms of anæmia are paleness of the visible mucous membranes, with often an unhealthy or slate-coloured appearance of that of the nasal cavity. In some instances a coppery appearance is witnessed; this is undoubtedly due to an altered condition of the globules, and a disordered state of the powers by which they are formed; the conjunctiva is pale, the mouth cool, and the tongue generally unnaturally soft. Young, growing animals, insufficiently fed and improperly cared for, exposed to the vicissitudes of the weather during the winter months, are reduced to an anæmic condition, manifested by emaciation, debility, and often a depraved appetite. When thus reduced, great care is to be taken that such are not suddenly turned into rich pastures, for it seems that the process of the formation and elaboration of the blood, and the organs concerned in it, are in such a state of disorder from long disuse, that the alimentary matters contained in luxuriant grass or nourishing food are transformed into an imperfect blood-plasma,—that is to

say, into blood abundant in quantity, but defective in quality, plasticity, and consistence, incapable of nourishing the tissues, and which ultimately becomes of itself diseased. From this cause—namely, the unwise and indiscriminate method in which young, rapidly growing, and imperfectly fed cattle are turned into rich pasturage in the spring, or put upon cake in the winter—gastric, blood, nervous, and other important diseases are caused, and the lives of valuable stock endangered. In horses I have repeatedly witnessed the anæmic condition associated with such extreme debility of the muscular system as to simulate spinal paralysis, and in some cases paralysis, from the pressure of serous fluid on the spinal cord, has been actually present; but in the horse, as in horned cattle, active signs of disease are not manifested until there has been a sudden change to a nourishing diet. Sheep fed on food containing an excess of moisture, and during long-continued wet weather, become anæmic and dropsical. During the winter of 1872-3 this condition was exceedingly prevalent owing to the inferiority of the grasses, the moist condition of the turnips, and the humidity of the atmosphere; for in many instances no organic disease or parasitic invasion was present.

The anæmic condition is announced by abnormal sounds in various parts of the vascular system. These are called venous murmurs, and are ascribed to the thin, watery blood running with great rapidity in the ill-filled vessels, and are heard at the breast, at the base of the jugulars more particularly, and are, according to Dr. Williams, of the nature of ripples, “the natural inequalities of the surface over which the current of blood passes being sufficient to occasion, in its dilute and diminished condition, vibrations and sonorous gushes, which would not occur in a fluid of greater density.”

The nutrition of the various structures of the body being dependent upon a healthy condition of the circulating fluid, it naturally follows that emaciation and debility are the results of anæmia. In none of the anæmic states, unless it be intensified by an excessive discharge, as in diarrhœa, is rapid emaciation so prominent a symptom as in that caused by the presence of *Bacillus mallei*, and in malarial affections.

The pulse in anæmia is feeble, thready, often jerky and irregular, and any sudden excitement produces a degree of palpitation of the heart, leading one to suppose that the organ is



hypertrophied, so loud, sharp, and knocking is the character of its contraction.

The contractile power of the muscles is generally much impaired, fatigue being soon induced; the bowels are often constipated, the constipation being due to a want of tone in the muscular coat.

Though the muscular system manifests great loss of power and tone, the nervous system seems to be exalted, excitement being induced by very trivial causes, but this seeming exaltation is in reality due to loss of strength and power. "The nervous system in the anæmic condition may be likened to a spring which originally was of a certain strength, requiring a certain impressing, and reacting with a corresponding force, but having become much weakened, is bent by much less force, and reacts also with much less. Mobility and debility may be said briefly to be the chief characteristics of the nervous actions in the anæmiated."—(Dr. HANDFIELD JONES.)

The digestive powers are feeble and imperfect, and colicky pains are induced by trivial causes; tympanitis is frequent, and results from the defective power of the muscular coat of the alimentary canal. The secretion of the gastric juice is also impaired, and results from the gastric follicles being insufficiently supplied with nutritive materials. This constitutes a further cause for the continuance and aggravation of the anæmic condition, and acts as a bar to healthy digestion, though the food supply be liberal and good, for healthy chyle cannot be formed to renovate the blood if the primary digestion be impaired. Most probably the accession of disease, when nutritious food is supplied to half-starved animals, is due more particularly to this cause.

Anasarcous swellings of the limbs is a common symptom of anæmia in the horse, but is rarely witnessed in horned cattle. In sheep, as already stated, dropsy of the cavities is frequently witnessed. Under the term "spontaneous anasarca," Mr. Brown of Melton-Mowbray has described the anæmic condition, with dropsy of the cavities, in the horse. Mr. Brown says the disease attacks one and two year old colts, grazing during the winter season in wet, poor moorland, and is very prevalent in Lincolnshire; the disorder being manifested by swelling of the legs, sheath, belly, and lips, and finally the eyelids, the breath and

excretions exhaling a peculiar odour. The animal turns sluggish and depressed, loathes its food, and seldom lies down; the respiration becoming accelerated and the pulse feeble. Loss of flesh, prostration of strength, short and difficult respiration, frequent and indistinct pulse, and finally diarrhoea, which, if once established, carries off the animal in despite of all remedies. The *post mortem* examination reveals yellowness and flaccidity of the muscular system, effusion into the areolar tissue, thickening of the pericardium and pleura, with effusion into their cavities, often so great as almost to cause collapse of the lungs and arrest of the heart's action; effusion into the omentum, mesentery, and peritoneum.—(See *Veterinarian*, 1832.)

The causes of anæmia being generally apparent, I need scarcely state that they must be removed, as a primary step in its successful treatment; and for this purpose, it was, or is, the custom in Lincolnshire, to take up young colts for the month of August, and feed them during that time exclusively on dry provender. When anæmia is due to the operation of a specific virus, as that of glanders, or when at any time its cause cannot be discovered, the employment of any mere remedial measures is of very doubtful utility. Again, the anæmic condition, which can be ascribed to well-known and preventible causes, may have existed so long, or operated so strongly on a constitution perhaps naturally weak, as to act as a determining cause of some cachectic condition such as tuberculosis, or to disease of the osseous system; or may have so impaired the action of the heart—the fibres of which have become organically altered, degenerated—as to prove fatal, notwithstanding all rational treatment. The treatment calculated to overcome anæmia consists in allowing nutritious diet, carefully selected, and of easy digestion: for horses—oats, beans, crushed linseed, the latter in small quantities, and good hay; for cattle and sheep—an allowance of oilcake in addition to their ordinary fodder, which should be of good quality, and sufficient but not over-abundant in quantity; in addition to which the salts of iron are to be given in small and repeated doses. If the digestion be weak and the appetite bad, the iron is to be withheld, the vegetable tonics and the mineral acids given. Should the appetite be depraved, or if there be an unnaturally acid condition of the juices of the stomach, manifested by a tendency on the part of the animal to lick the walls and grind its teeth, with sourness of the mouth and fætor of the fæces, the alkalies are to be com-

bined with the vegetable bitters. For the constipation, which is due to debility of the muscular walls, *nux vomica* is very useful, mashes being now and then allowed. To the food above prescribed, it may be necessary in aggravated cases to allow the animal to have a quantity of milk twice a day—say one gallon of skimmed milk night and morning, mixed with oatmeal or porridge; this it will usually become very fond of in a day or two. The system of forcing food, by horning it, upon animals, is not to be recommended; for what an animal does not eat spontaneously is not digested, but disorders the digestive system, and destroys any little appetite that may possibly remain.

Horses, during the spring and autumn, when casting their coats, suffer more or less from anæmia and debility, and are incapable of performing the same amount of work as at other seasons of the year. A little rest and gentle treatment at this period often prevent the accession of disease. Again, horses whose coats are very long, if kept in stables of even ordinary warmth, suffer from anæmia, with anasarca, and even dropsy of the serous cavities, which will increase notwithstanding the most powerful tonics and the most nutritious food, until their heavy coats are removed by clipping. I have seen the lives of many valuable horses saved by timely clipping.

In all anæmic cases the secretory organs—the kidneys, skin, &c.—are very apt to become torpid; the urine is scantily secreted, the skin dry and harsh, the animal hide-bound, as it is termed. These complications must not be overlooked, or the blood will become loaded with effete materials. To stimulate the kidneys the spirits of nitrous ether may be given occasionally, and to excite the skin nothing is to be compared to good grooming, particularly damp wiping the whole surface of the body. Warmth, comfort, gentle exercise, and pure air all play an important part in the treatment, and should by no means be neglected.

The changes and alterations in the several constituents of the anæmic blood are briefly as follows:—(1.) The red corpuscles are remarkably diminished; they have been known in the human being to sink extremely low in number; they also contain less hæmatin, and are somewhat paler than those of healthy blood. (2.) The amount of white corpuscles is usually increased. (3.) The coagulability is often very low, showing a want of fibrin or of the fibrin ferment, but this is best seen in

that form of anæmia, occurring in malarial infection and piroplasmosis, when there is mechanical destruction of red blood cells. In some cases its quantity is notably increased, particularly if inflammation of any organ is present. The marked tendency to coagulation noticed in the exudate (pleural, peritoneal, mucous, &c.) in inflammatory affections of these tissues, must not lead one to imagine that there is any increased tendency to this change in the blood itself. As Kitt points out, all exudations contain a great deal of the fibrin-forming element, and the ferment is supplied by the changes taking place in the proliferated cells of the affected surfaces. As a rule, in all acute inflammatory diseases the blood itself shows a decided disinclination to normal clotting, and this is equally noticeable in many chronic wasting diseases. (4.) The solids of the serum are not found much altered. (5.) The quantity of plasma in the blood in cases of anæmia is seldom increased, but owing to the marked diminution in the number of red corpuscles, there is an apparent increase, both macro- and microscopically.

#### LEUKÆMIA OR LEUCOCYTHEMIA

Is a condition of the blood characterised by a large increase in the number of the white corpuscles, associated with a new formation of lymphatic tissue (lymphadenoma) in the lymphatic glands, spleen, and other organs; but the association of leukæmia with lymphadenoma is not constant.

An increase of white corpuscles is also witnessed in some cases of farcy and glanders; but the true nature of leukæmia seems to depend more upon an inability of the normoblasts or nucleated red cells to form red blood corpuscles, than upon any actual growth of lymphatic tissue.

#### POLYÆMIA, PLETHORA.

The possibility of there being at any time an increased quantity of blood in the animal body—plethora—is denied by some observers. The condition, however, is recognised by veterinarians as one of not infrequent occurrence, more especially in young fast-thriving animals, when fed upon very highly

nutritious food. The occurrence of a general plethoric condition is also recognised by Sir Thomas Watson, who says—“Perhaps it may not be so obvious that the whole quantity of blood throughout the body is sometimes in excess. . . . Full living and a sedentary life are causes likely to occasion general plethora, and they do occasion it. The full diet, so long as the digestive powers are perfect, provides more chyle, conducts into the blood a larger quantity of its proper pabulum. . . . Their entire vascular system is preternaturally distended. If you open a vein you find that they [persons in a plethoric condition] bear a copious abstraction of blood without fainting, and are even refreshed by it; and the blood drawn separates into a large and firm mass of coagulum, with but little serum. Keeping to the nomenclature we have already employed, we might say that there is here *hypertrophy* of the blood.”

The treatment of these cases is sufficiently obvious—abstraction of blood, purgatives, a restricted diet, and increased exercise.

## CHAPTER IX.

### PATHOLOGY—(*continued*).

#### CHANGES IN THE BLOOD—(*continued*).

##### DEFECTIVE EXCRETION.

SIR THOMAS WATSON, in his graphic language, says that “the animal fluids are—the blood, the fluids that enter the blood, and the fluids that proceed from the blood.

“The fluids that enter the blood are of two kinds—

“1. Those by which it is renewed and enriched.

“2. Those which enter it in order that they may be conveyed out of the body.”

A defective condition of the fluids which enter the blood to renew and enrich it—namely, the chyle—produces the condition already described—anæmia. A modified or altered condition of the chyle—that is to say, chyle imperfectly developed, containing abnormal constituents, products of imperfect digestion or elaboration, or directly derived from the ingesta—is a frequent source of alteration in the blood and of disease.

The fluids which enter the blood for the purpose of being conveyed out of it are the products of the metamorphosis of the chyle and tissue, accidental materials contained in the food, products of imperfect digestion, superabundant nutritive elements unappropriated by the tissue cells, and undergoing degradation within the blood-vessels, or in organs such as the liver. For the removal of these effete materials the organs of excretion are provided. When the organs of excretion are disordered or diseased, it follows that such products accumulate in the blood, contaminate and render it unfit for the proper nourishment of the body.

The most remarkable and perhaps the most important change in the blood from defective excretion is that produced by arrested or deficient secretion of urine. In some experiments performed by MM. Prevost and Dumas, who removed the kidneys of dogs and cats, on the third day after the operation vomiting came on, with diarrhoea of a copious brown liquid containing ammonia, from transformation of the urea, eliminated from the intestinal mucous membrane; fever, with heat sometimes as high as  $110^{\circ}$ , sometimes as low as  $92^{\circ}$ ; small and very frequent pulse, laboured breathing, and death, from the fifth to the ninth day.

The *post mortem* appearances were, effusion of serum in the brain, much mucus in the bronchial tubes, bilious fluid and fæces in the intestines, an appearance of inflammation of the liver, great contraction of the urinary bladder, the blood more watery than usual, and containing urea; five ounces of blood of a dog yielding twenty grains of urea, and two ounces of cat's blood ten grains.

Defective secretion of urine, resulting from disease of the kidneys, induces symptoms very similar to the above, differing in degree and intensity according to the extent of the kidney disease. Thus, in acute nephritis, or in rapid degeneration of the kidneys, such an amount of urea and other effete urinary matters may collect in the blood as to cause convulsions, delirium, suffocative discharge of mucus from the bronchi, diarrhoea, and serous effusion into the cavities and areolar tissue. In subacute cases dropsy gradually ensues, the animal finally becoming cachectic. All these effects may be traced to excrementitious materials retained in the blood, especially urea, which if in great amount acts as a narcotic poison, in smaller, as an irritant, inducing inflammations in various membranes; in the horse, more especially in the membranes of the spinal cord, and the neurilemma of the great nerves given off from the lumbosacral plexus.

#### TRANSFORMATION OF CHYLE AND OF THE TISSUES.

The blood is kept in its normal or healthy condition by being supplied with new elements derivable from the chyle, and by having the products of the metamorphosis of the tissue and all

its effete constituents removed by the various excretory organs. When any of these conditions are absent, or when, as sometimes happens, the waste of tissue is so rapid as to preclude the elimination of all its products, a mal-condition of the blood-mass is induced, which may lead to grave local complications and diseases of important organs.

Some chemists hold the opinion that urea more especially is derivable from waste of tissue only, and not from any change or metamorphosis in the chyle or blood. I shall, however, endeavour to prove that an uræmic condition of the blood may be caused by chyle imperfect in quality or excessive in quantity; the imperfection of the quality being the result of disordered digestion, or of the ingestion of food unfitted for healthy chylification; and excessive in quantity from the animal being fed upon aliment too rich for its requirements. In order to understand this, however, the student must be made still further acquainted with the process of nutrition, and this may be divided into—(1.) The introduction into the stomach and alimentary canal of the food to be digested: (2.) The formation of the chyle and blood, and the changes which the latter undergoes in the lungs: (3.) The passage of nutritive plasma from the blood to be transformed into tissues: (4.) The metamorphosis and re-absorption of the tissues: (5.) The excretion of these effete materials from the body by the various excretory organs.

I shall, however, confine myself, very briefly, first, to the digestion of the food, and the formation of the chyle; and second, to the changes and transformation of the chyle and blood in the vessels.

All kinds of food are resolvable into carbon, hydrogen, oxygen, nitrogen, and mineral constituents; and food to maintain the animal body in a state of health must contain all these various substances in due and proper proportions, and in quantities depending upon its necessities and wants under the circumstances in which it is placed, the amount of work it is compelled to perform, the amount of air it breathes, and upon various peculiarities affecting its powers of digestion, whether the animal be carnivorous, omnivorous, or herbivorous.

It is immaterial to what natural order the animal may belong, the proximate chemical principles required for its nourishment are the nitrogenous—albuminous, non-nitrogenous—starch, gum,



sugar, oil, and the mineral salts, more particularly the phosphate of lime and the chloride of sodium, with iron, potash, &c.; and it has been proved that all nutritive food must contain the three principles, and that the absence of any one of them induces starvation and death. It is also proved that the chemical constitution of plants and animals is nearly identical, and hence food derived from plants contains the substances of which the animal body is made, and by which it is nourished. Some animals are carnivorous, and must be maintained on flesh. In others life is supported by plants and grain. The digestive organs differ somewhat in these; but the digestion—that is to say, the ultimate end of the digestive process—is the same in all, namely, the formation of a whitish emulsion, the chyle, which is absorbed from the intestinal canal, and conveyed into the general circulation, there to be transmitted to all parts of the body, giving to each materials which it may at the time stand in need of for its development, growth, and vitality. In all animals water is also necessary, not only as a diluent, but as forming a component part of the blood and tissues.

It must be understood that mere nitrogenous, non-nitrogenous, and saline materials of food will not serve the purposes of healthy nutrition; they must be blended and united to form the kind of food natural to each class of animal, and capable of being finally converted into albumen, fat, and salts, as all these exist in every tissue; the fibrous and muscular organs containing much albumen, the glands and areolar tissue fat, and the bones mineral matters.

The quantity of food required is greatly influenced by the weather. If cold and condensed, the air contains more oxygen, which, rapidly uniting with the tissues for the purpose of maintaining the normal standard of animal heat, the body demands more nourishment to prevent emaciation. Again, work or severe bodily exercise causes waste of tissue, and at the same time, if not too severe, stimulates the appetite, and improves the powers of digestion. The demand for more food, the increased appetite, and the exalted digestive powers being necessary to the wellbeing of the animal, as compensating agents, by which more food is partaken of and digested, in order to make up the waste of tissue induced by the bodily exercise, a more or less even balance is thus kept up between the secondary digestion,

or destructive process, and the primary digestion, or compensating process.

It is very true that a healthy animal will retain its appetite and be capable of partaking of food and digesting it for a considerable time without exercise, and, under certain circumstances, which sometimes seem inexplicable, remain in health. Generally, however, the excessive amount of nutriment is stored up amongst the fatty tissues in various organs; and the storage of fat may so far be looked upon as an act of excretion, bearing out the proposition of Treviranus, that "each single part of the body, in respect to its nutrition, stands to the whole body in the relation of an excreted substance;" or, in other words, every part and tissue of the body, by taking from the blood the peculiar substance of which it is made, or which it can accommodate and store for future use, acts as an excretory organ, inasmuch as it removes from the blood that which, if retained, would be injurious to the rest of the body.

In many cases, however, this act of accommodation, as it may be termed, is insufficient; and in animals fed upon rich food, particularly nitrogenous food, the blood-mass becomes so impure, from the presence of effete materials, that the whole body is poisoned. For example—and this not at all uncommon—a horse is kept in the stable for some days, perhaps three or four days, and fed upon its usual liberal allowance of food, consisting of hay, corn, and water. When put to its work it usually commences its labour with the greatest spirit and animation, every nerve and muscle being, as it were, in the highest state of functional perfection; but before it has proceeded more than a few miles on the journey, very often before it has gone a mile, it suddenly staggers and falls paralysed. Sometimes it may be able to rise and walk slowly to its stable; sometimes it never rises again. Shortly after the attack it is observed to pass large quantities of a dark coffee-coloured urine, so dark, indeed, as to lead many observers to suppose that it is blood. Careful observations have, however, enabled me to determine that such urine contains no blood, and little or no albumen; but that the darkness of its colour depends upon an excessive quantity of colouring matter, urea, and probably other products of the degradation of the nutrient materials which had accumulated in the blood-mass during the period the animal was at rest.

It has been maintained by some writers that urea is a product formed in the kidneys, and by others that it is an oxidized albuminous material, due to the degradation of muscular tissue, brought about by muscular exertion only, and merely excreted by the kidneys.

Without a doubt urea is an oxidized or degenerated condition of albumen, but the occurrence of the immense quantity of it in conditions of the animal body opposed to that of tissue change, I think, disposes of the idea that urea is due only to metamorphosis of tissue, and supports the theory that it is formed in the blood itself, or in the liver, as advocated by Meissner.

The rules for the proper treatment of disease depending on blood loaded with effete materials are—1st. To clear the intestinal canal of the materials with which it may be loaded, and from which additional matter may be absorbed and conveyed to the blood: 2nd. To stimulate the powers of the excretory organs, kidneys, and skin: and 3rd. To attend to the comfort of the patient, and particularly to supply it with abundance of water, which not only allays the thirst, but dilutes the blood and stimulates excretion.

#### MELLITÆMIA.

The sugar in the blood is increased in certain diseased conditions—*diabetes mellitus* and in some cases of azoturia in the horse. It is then partly excreted by the kidneys. True diabetes mellitus is but rarely witnessed in horses, and perhaps never in cattle and sheep, but is not uncommon in dogs; and to replace the loss of sugar thus given there is an increased metabolism of the nitrogenous structures, and consequently the amount of urea is increased. There is loss of flesh, the glands atrophy, and the crystalline lens becomes opaque, and there being an increased amount of sugar in the humors of the eye, the water of the lens is extracted (Osmosis).

## CHAPTER X.

### PATHOLOGY—*continued.*

#### CHANGES IN THE BLOOD—*continued.*

##### HYPERÆMIA OR CONGESTION.

THE liability of the blood to be variously altered and modified having been described in the preceding chapter, it now becomes necessary to enter into a consideration of certain changes which may occur in its circulation, arising from an alteration in the condition and properties of the blood-vessels, and causing an interference with its natural flow.

By hyperæmia is meant an excess of blood in the vessels of a part or organ of the body. It is of two kinds, namely, active or arterial, and mechanical or venous.

Active hyperæmia is an excess of blood in the arteries, with, most commonly, an increased rapidity of the flow, and arises from increased blood pressure and from diminished arterial resistance.

Hyperæmia from increased blood pressure occurs from interruption of the main current of blood in any particular part, owing to which increased pressure is thrown upon the collateral vessels, which thus become dilated, the amount of blood in them being increased, and its flow accelerated. This kind of congestion is seen after the main current of a part has been obstructed from any cause, and is known as *collateral hyperæmia*.

Congestion of the vessels of one part from an obstruction to the flow of blood in another, is also well exemplified in congestion of the internal organs from contraction of the superficial capillaries, so often produced by exposure to cold.

In some instances, the intropulsive operation of cold is so great that the internal capillaries become loaded with blood to such

an extent that their walls give way, and the blood is extravasated into the surrounding tissue. In this manner apoplexy of the lungs is brought about, and so great is the extravasation in some rare instances, that the blood has found its way into the bronchi and trachea, and has been finally expelled through the nostrils. The local constricting effect of cold is best witnessed upon the human body, more especially in persons of weak circulation, in whom, very often after washing or bathing in cold water, the fingers are quite bloodless, pale, and numb. If we reason upon this circumstance, and bring such reasoning to bear upon our own subject, can we wonder that exposure to external cold is followed by internal congestions.

In many cases, however, nature provides a guard to the influence of cold upon the skin, by furnishing animals with coats of hair or wool suitable for their several necessities, and it is only in animals which are *casually* exposed to such cold that we witness its effect in causing internal congestions.

Congestion from diminished resistance arises from atony of the wall of the vessels. This atony, loss of power, or relaxation of the vessels, is generally due to paralysis of their muscular walls, and most commonly occurs in vessels least provided with muscular fibres, that is, in the capillaries and veins of organs easily distended, and which are composed of much areolar tissue. This paralysis of the muscular walls may be due to an influence operating upon the vaso-motor nerves, or to irritation of a sensory nerve. Pressure upon the great sympathetic nerve in the neck causes active congestion of the head and neck, and most probably the congestion of the brain, redness of the conjunctivæ, suffusion of tears, and other signs of fulness of the vessels of the facio-cranial region, with the coma and delirium which are sometimes associated with gastric or intestinal disease, are due to pressure upon the cardiac branches of the great sympathetic or ganglionic nerve. It is very true that some emotions are attended with hyperæmia; and it may be said that the meningeal or cerebral congestion of parturient apoplexy is due to some sympathetic influence which the stomach has upon the brain. The question need not be discussed here, as it will again come under consideration; and let it suffice us for the present to know that disorder of the stomach is capable of producing congestion of the brain.

In hemiplegia, a disease rarely witnessed in the horse, but to which horned cattle are liable, congestion of the vessels of the paralysed side is a prominent symptom.

Atony of the arterial walls may be due to degeneration of their muscular and internal coats. It is most commonly the cause of the various congestions seen in old animals.

Mechanical congestion occurs chiefly in the veins, and is produced by anything which interferes with the flow of the blood through them, such as obstruction of the vein leading from a part, arising from pressure upon its walls, or the pressure of a thrombus or clot within its canal. Thus congestion may be induced by the pressure of a tight bandage; and again, congestion of the portal vein is caused by cirrhosis or other disease of the liver, which causes pressure upon its venous branches. Disease of the valves of the heart produces fulness of both the pulmonary and systemic veins. When the incompetency is limited to the mitral valve, the congestion is mostly limited to the lungs, while insufficiency of the tricuspid causes congestion of the systemic veins. The character of congestion beginning in the veins is that the veins as well as the capillaries are distended, giving the part an arborescent appearance, which has a much more permanent character than congestion beginning at the arteries. Arterial congestion, especially that of an active type, very often disappears shortly after death by the *post mortem* contraction of the vessels.

In addition to mechanical obstruction to the flow of blood, there are various and important causes of venous congestions, such as diminished cardiac power, gravitation, an altered condition of the blood, and defective secretions.

1. *Diminished cardiac power*.—The contractile power of the heart becomes diminished in many diseases, and death may result, not so much from the original disease from which the animal has been suffering, as from congestion of some vital organ, more especially the lungs or the brain, arising from the inability of the heart to propel the blood through the vessels of such organ. The example most familiar to the veterinarian of this form of congestion is acute congestion of the lungs caused by over-exertion. Many instances of this occur in the hunting-field during severe runs; horses out of condition, and unfit for very prolonged and severe galloping, falling down and rapidly sinking

from this cause. This form of congestion becomes aggravated by the blood remaining unchanged, for it is found that when the blood is not properly changed in the lungs, its passage through them is partially impeded, that it accumulates in them, in the right side of the heart, and in the veins generally.

2. *Gravitation*.—The veins of the extremities and other depending parts of the body are congested from this cause. It indicates an atonic condition of the system generally, with diminished cardiac force; and is commonly associated with all the exhausting diseases of the horse, more especially if the animal is not exercised. Congestion from gravitation is also caused in many healthy horses by want of exercise. Indeed, a due amount of exercise, when in a condition to take it, seems to be of greater importance to the horse than to horned cattle. The cow or ox will remain in the stall for weeks, or even months, tied in one position and receiving no exercise, but the limbs never swell, and there are seldom any signs of venous congestions; not so with the great majority of horses. Now and then, however, a horse is met with whose limbs never swell from want of exercise, but as a rule horses when not exercised suffer from swellings of those depending parts of the body farthest removed from the centre of the circulation. Other horses habitually suffer from swelled legs, no matter how carefully and properly they are fed and worked. The debility produced by the process of “casting the coat” causes congestion of the vessels of the depending parts.

A very remarkable example of the effects of gravitation in producing congestion used to be frequently witnessed in the days when bleeding was so commonly performed, and when inflammation of the jugular vein was more frequently met with than now.

The loss of a jugular from the formation of a thrombus within it, and consequent obliteration of its canal, did not seem to interfere with the cervical circulation until the animal was turned out to grass; but immediately this was done, the animal's head would commence to swell to a most alarming extent, proving that, although the collateral circulation was sufficient, so long as the horse was fed from the rack or manger, it was quite insufficient when the head was depressed whilst grazing.

3. *Altered conditions of the blood*.—Many congestive diseases,

more especially those of horned cattle and sheep, are caused by an altered condition of the blood. How this determines a congestion of an organ or a system of organs it is difficult to understand; but such diseases as apoplexy of the spleen, quarter-ill, and other anthracoid diseases manifested by extensive congestions and extravasations, are all due primarily to blood alterations, the lesions of organs being secondary to and depending upon the blood disease. A thin watery state of the blood, as well as the presence of specific organisms, such as piroplasms and trypanosomes, have also in many cases a tendency to cause congestions of organs.

4. *Defective secretion*, either from an organ or a secretory surface, is a cause of congestion of such an organ or surface; and it is found that the means which increase the secretion will often remove the congestion. It must, however, be borne in mind that diminished secretion is usually the result of the congestion, and that the means which have the power of exciting the secretion of such a part very often, in virtue of their irritating effects upon it, increase the congestion, or, what is perhaps of greater moment, transform a mere congested condition into that of inflammation.

Many writers seem to confound congestion with inflammation, and we continually see slight inflammations described as congestions. It appears to me that a distinction should be made between the two conditions, and that a congestion should be defined to be a fulness of the vessels of a part or parts arising from various causes, without the occurrence of any primary important perversion of the nutrition of that part; and that inflammation is a perverted condition of the nutritive function of a part, accompanied in the majority of cases by a congested condition of its vessels; such congestion, however, not being necessary to the inflammatory process, for it is found that inflammation involves the non-vascular as well as the vascular structures of the animal body.

The results of mechanical congestion are transudation of serum, hæmorrhage, thrombosis, and gangrene; and of active hyperæmia, increased redness, elevation of temperature, and a sense of throbbing, succeeded, if the congestion continues, by enlargement of the small arteries and thickening of their walls; function being in some cases exalted, more especially if the



congestion be in a nervous centre, whilst in other instances function becomes diminished. Thus a long-continued congestion of a secretory organ arrests, or very considerably diminishes, the function of that organ.

The congestion or fulness of the vessels is somewhat relieved by transudation of plasma into the tissues, or into cavities, constituting cedema, anasarca, and dropsical effusion. For this reason the limbs swell when their vessels are congested from debility or want of exercise; and the peritoneal cavity becomes filled with fluid, constituting ascites, when the portal veins are in any way obstructed. The transuded serum generally differs from that of the blood, in containing less of the solid blood constituents and more water; it has also a lower specific gravity. If exposed to the air it will sometimes coagulate, forming rather a gelatinous mass than a firm fibrinous clot.

When the obstruction is very great the exudate is of a more coagulable nature, due to the presence of the fibrin ferment components, and the blood globules escape through the walls of the capillaries without rupture. This fact was discovered by Cohnheim, who, by applying a ligature to the femoral vein of the frog, observed, when the congestion of the vessels became very great, that the red corpuscles passed through the capillary walls, and in so doing assumed an hour-glass shape, or became constricted in their middle. This fact explains to some extent how, in some cases of purpura, transudation of blood takes place into the tissues without rupture of the vessels.

Congestion from obstruction producing gangrene is seen in strangulated hernia, and in invagination of a portion of the intestines. Gangrene from obstruction is always of the moist kind, whereas that from arterial incompetency is of the dry kind. In the one the blood supply is scarcely or not at all interfered with, but the return of that blood is arrested, stasis is produced, the blood thus imprisoned transudes into the tissues, and, rapidly dying and decomposing, adds to the rapidity of the death and decomposition of adjoining structures. In the other, or gangrene from want of blood, the part turns pale and dry, shrinks, becomes mummified, or crumbles into fragments.

The occurrence of thrombosis, or the formation of clots in the congested vessels, results from the blood coagulating within them during life, and may arise in congestion of any part, and

from any of the causes already enumerated. When caused by direct pressure, the coagulation commences at the point where the obstructing cause comes into contact with the vessel, and extends as far as the collateral branches; when from debility, diminished cardiac power, or gravitation, the coagulation commences first behind the flaps of the valves, and appears to be owing to the force of the current being insufficient to completely open the valves. The blood thus allowed to lodge and stagnate coagulates and forms a thrombus or clot, which may finally obstruct the whole venous channel as far as the next large collateral branch.

*Treatment of congestion.*—The most important means for the removal of congestion are those which tend to remove its cause; and if the causes already enumerated are borne in mind, the practitioner will at least have some guide to the proper line of treatment. When the congestion is due to the influence of cold upon the surface, clearly the first step is to restore the circulation of the skin by warmth, friction, and by the administration of remedies, such as the spirits of nitrous ether, which are known to promote diaphoresis. If the congestion be due to diminished cardiac power, or debility of the vessels—and this is often induced by want of exercise—the lost power and tone must be restored by good food, pure air, regular and moderate exercise, and tonics. If the debility of the vessels be very great and obstinate, the effects of ergotine may be tried. This remedy acts by producing contraction of the blood-vessels; it may be very conveniently used hypodermically. The effects of bandaging, in supporting the weakened vessels of congested limbs, should not be lost sight of, nor its application neglected. In many instances a watery condition of the blood is associated with debility of the circulatory apparatus: in such cases tonic remedies combined with diuretics act most favourably.

In severe internal congestions, where not only the organ chiefly affected is filled with blood, but the right side of the heart and the great veins also, the necessity of affording mechanical relief becomes a serious question, and very often an urgent necessity. It is very true that severe internal congestions, more especially those of the lungs and brain, are generally associated with greatly diminished cardiac power, feeble pulse, and nervous prostration; and if we were to admit what is so commonly in-

sisted upon, that the course of treatment is to be guided by the condition of the pulse, then the withdrawal of blood would be contra-indicated in every instance. But practitioners who have thought over the matter know full well that a moderate bleeding is followed by a diminished acceleration of the pulse, and improvement in its tone, and that many congestions are immediately relieved by such procedure. The explanation is not difficult. In cerebral congestions the diminished cardiac power and feebleness of the pulsations are caused by the pressure of the congested vessels upon the brain substance: in many instances this pressure is increased by the transudation of serum from such congested vessels. By diminishing the brain pressure by the abstraction of blood, the cause of the debility is at once removed. The heart is now able to beat fully and strongly, and thus a small, feeble, or even thready pulse is changed into a full, soft pulsation. The variety of opinions held as to the possibility of causing an alteration of the fluids within the cranium by bleeding or otherwise, need not be discussed at present. My own experience leads me to the conclusion that a comatose condition produced by cerebral or cerebro-meningeal congestion is often removed, the pulse being at the same time improved in tone, by a moderate bleeding. Pulmonary congestion and the feebleness of the heart's action—the “oppressed action” as it has been termed—are aggravated by the right side of the heart and the great veins being choked with blood. In this instance, it is true, the withdrawal of blood does not relieve by removing the primary cause—that is, the congestion of the pulmonary capillaries—but, a most important complication, it diminishes the supply of blood to the already overloaded heart, and through it to the already congested vessels. Bearing this in mind, the rapidity with which the withdrawal of a large stream of blood produces an impression will be understood; indeed, it can most truly be stated that the relief afforded does not depend so much upon the *quantity* withdrawn, as upon the rapidity of the withdrawal. For example, six or eight quarts of blood might be abstracted in a small or trickling stream, and afford no relief, but, on the contrary, do much harm, by causing debility or exhaustion; but a smaller quantity, if withdrawn rapidly, may be followed by a marked remission of all the distressing symptoms. In the one case, the blood supply to

the heart within a given time has scarcely been interfered with, while, in the other, the quantity returned by the veins has been most sensibly diminished, time has been afforded to the congested vessels to become to some degree emptied, and regain their lost tone, and for the circulation generally to be restored to its equilibrium. In these congestions the administration of diffusible stimulants after the bleeding will still further restore the lost power of the propulsive force and of the debilitated walls of the vessels; and, in virtue of their exalting action, will diminish the internal congestion, by causing an increased circulation of the vessels of the skin and excretory organs.

Local exudations, especially those seen in horses' legs and such conditions as chilblains in human beings, are often the result of a loss in coagulability of the blood. It has been ascertained that the blood under normal conditions has a fixed time to coagulate when withdrawn from the body. If this time is exceeded, the blood is low in coagulability; if it take less than the normal time, it has a high coagulability. These facts have latterly greatly assisted in the treatment of diseases. Wright has given a very happy metaphor in this respect. He illustrates it by showing that if a canvas hose-pipe has thick fluid gelatine passed through it, the force required to cause any exudation through the canvas must be very great. If, however, the gelatine be very fluid, the force required will be very much less. It is the same with the blood. If a horse's blood is low in coagulability, that horse will suffer from oedematous legs, and its resultant evils, if the vessels are deprived of the help which they receive from the pressure exerted by muscles during exercise. If, however, the coagulability be normal, there is not this tendency. It is well, therefore, to ascertain the coagulability by taking a fixed quantity of blood in a pipette, and testing its time of clotting on a piece of blotting-paper, which should be in two minutes. Such drugs as lactate of calcium will accentuate clotting and citrate of soda will delay it.

#### LIPÆMIA, OR FATTY BLOOD.

In February, 1882, I was consulted by a gentleman living in the West Highlands regarding a fatal disease which had appeared amongst his ewes.

The history of the outbreak was very meagre, and was from the farm manager, who stated—

“The appearance when first attacked, or rather when we notice them, is that they do not follow the flock, and, when walking, propping their legs as appearing to keep them from falling, which they often do, always going down head first.

“Their droppings not at all natural, and mixed with a slimy mixture of blood. They live from three to four days after we detect them ill.

“Their food for the last three months, turnips carted out on grass land, with an allowance of half a pound of Indian corn and cotton cake mixed per day.

“I may add that they were turned into the turnip fields to pick up broken and small turnips, but were only there about three weeks, during which time they had full turnips, and looked the very picture of health.

“Mostly all the dead ewes when opened have shown a great deficiency of blood.”

The proprietor in a short note informed me that “the only treatment that has been attempted is giving a dose of castor oil, and that some of those showing symptoms of disease improved since the giving of cotton cake was discontinued.”

These letters were accompanied by the carcase of a sheep of the Shropshire Down breed in an advanced state of decomposition.

On a *post mortem* examination being made the whole body was found to be loaded with fat; that of the abdominal parietes being several inches in thickness; the sublumbar region had many pounds of fat in it, and here it was found that the interior of the fatty masses had undergone crystallisation; well-formed crystals of stearine and margarine being found in the midst of a dry kind of *débris*.

The structures of the liver and of the heart were not only thoroughly infiltrated with oil globules, but had undergone fatty degeneration to an advanced degree; the lungs were congested, and the large pulmonary vessels filled with thrombi; the cardiac ventricles were quite full of blood, showing that death had been induced by *æsthenia* and pulmonary congestion.

Microscopically examined, the blood (which was pale in colour when seen with the naked eye) from the heart, pulmonary vessels,

and from other parts, was found to be loaded with oil globules, as represented in the drawing, which was made on the spot.

It will be seen that the oil globules vary in size, some being smaller, some much larger than the red blood corpuscles. It was unfortunately, however, impossible to prepare microscopic slides

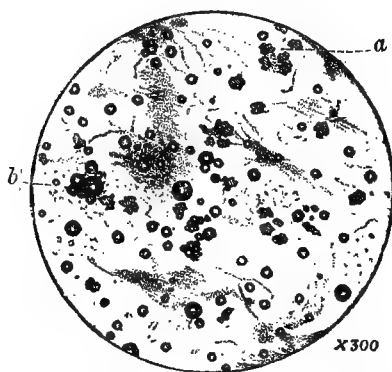


FIG. 9 A.—(a) Blood corpuscles altered in shape. (b) Oil globules, isolated and in groups, scattered about the field.

of the blood and tissue owing to the advancing decomposition ; but notwithstanding this, the fat gave the usual reactions with perosmic acid and other staining materials.

I am not aware that this disease has ever been observed by any veterinary writer, and it was only in 1874 that Professor Kussmaul first drew attention to it in the *Deutsches Archiv für Klinische*, as causing a remarkable mode of death in diabetes mellitus of the human being ; the fatal symptoms consisting in a peculiar kind of dyspnoea, which after a time was accompanied by and ended in coma.

In 1879, the late Professor Sanders, and D. J. Hamilton, M.B., Edinburgh, investigated two cases after death from diabetes, and found a condition of the blood similar to that described above.

Death from lipæmia has also been observed in cats which have suffered from exhaustive diseases, accompanied by great emaciation, but no cases have hitherto been recorded in which this condition of the blood has accompanied obesity of the body generally.

Fatty embolism of the pulmonary artery is a cause of death in some cases of compound fracture of the long bones in man, and is accounted for by the fact that the medulla of the osseous canal gains entrance into the ruptured blood-vessels, and is thus mixed up with the general circulation, becoming arrested in its passage through the pulmonary vessels, and there forming a block to the pulmonary circulation, which finally effectually stops the aëration of the blood, and causes death by apnoea.

On comparing the fat condition of the body of this sheep to the emaciation of diabetic subjects, one cannot help being struck with the contrast between the two extremes. It is certainly difficult to answer the question, how is the fat produced in the blood of diabetic patients? But in this sheep the conclusion seems self-evident: that we had an animal which (along with others) had been fed up to such a point that it could no longer either assimilate, utilise, or excrete the highly nutritious matters contained in its food.

The Shropshire Down breed of sheep are noted for their fattening properties, and in this case we had an example of an animal having been fed beyond the point of perfection. The winter of 1881-2 was a remarkably open one; grass was plentiful throughout the season, and, in addition to an abundance of grass, these sheep had a full allowance of turnips, besides corn and cotton cake. This rich food, the quantity of which, however, does not strike one as being outrageously great, given in addition to the turnips and grass to animals whose fattening qualities is so well known, was evidently too much. After feeding upon it, the sheep would have little or no inclination to roam about; indeed, grass being abundant, there would be no necessity for them to travel far in search of food; fat was consequently so rapidly formed that all the tissues of the body became loaded with it, and finally the blood, where it caused an obstruction to the circulation, and thus induced death.

#### SEPTICÆMIA—PROGRESSIVE GANGRENE.

*Definition.*—A condition produced by the absorption or introduction of septic matter into the system, characterised by great prostration, metastatic lameness, colicky pains, with purging, the fæces being foetid, foetor of the breath, and

rapid emaciation in the horse. In horned cattle and sheep it is generally seen after parturition, and manifested by great prostration, collapse, fluttering pulse, expulsion of a coffee coloured fluid from the uterus, violent straining, purging, and great restlessness; also known as braxy, affecting sheep in their first year (hoggs), and occurring in the late autumn and early winter months, especially when the weather is cold, and the grass covered with hoar frost. It is also very destructive to lambs, and to calves a few days old.

These varying forms of septicæmia will be found described under various heads in the context of this work, as well as in the *Principles and Practice of Veterinary Surgery*.

THE ARTIFICIAL INDUCTION OF SEPTICÆMIA has been the subject of various experiments—first of all by M. Davaine, and afterwards by M. Bouley, Director of the Alfort Veterinary School. The experiments were as follows:—Six watch-glasses were placed on a table. Into the first 100 drops of water were placed, and to these was added one drop of blood from a septicæmic rabbit; the whole was stirred so as to produce a solution of  $\frac{1}{100}$ . One drop of this was placed in the second glass containing 100 drops of water, and a dilution of  $\frac{1}{10,000}$  produced. In the third glass, a drop from the second gave a dilution of  $\frac{1}{1,000,000}$ . In the fourth glass a drop of third dilution added to the 100 drops of water produced a dilution of  $\frac{1}{100,000,000}$ . In the fifth glass, similarly treated, there was a dilution of  $\frac{1}{10,000,000,000}$ ; and in the sixth a drop of the dilution gave a trillionth ( $\frac{1}{1,000,000,000,000}$ ). Matters being so disposed, four rabbits were inoculated respectively with the first, second, third, and fourth dilutions, a horse with the second, and a guinea-pig with the first. Next day all the rabbits were dead, the guinea-pig fell ill, but recovered, and the horse sustained no harm. M. Bouley communicated an account to the Académie de Médecine of numerous experiments he had made confirming the above, and stated that dogs can also be killed by the septicæmic virus derived from the rabbit, and that such virus derived from the horse possesses much less virulence than that furnished by the rabbit.

I have repeatedly experimented on birds and rabbits with the undiluted blood of pyæmic—septicæmic (Vogel)—rabbits and birds, and have always induced death, either by a rapid



corruption of the whole blood-mass, from which the animals have died in a few hours, or, resisting the first effects of the morbid inoculated material, from pyæmia with abscesses in various internal organs.

Selecting mice as being specially adapted for experiments on infective diseases, Koch has made some remarkable discoveries pointing to the conclusion that death may be induced by (*a*) a soluble poison, "Sepsin;" by (*b*) a true septicæmia induced by the multiplication of the bacteria introduced by the inoculation; and by (*c*) a progressive destruction of tissue (gangrene), commencing at the point of inoculation and spreading rapidly to adjacent parts.

Blood or meat infusion, which have putrefied for a short time, act more injuriously than when putrefaction has extended over a longer period, and five drops of such blood, if injected under the skin of the back of a mouse, kills it within a short time. There are marked symptoms of illness immediately after the injection. The mouse becomes restless, runs about constantly, but showing great weakness and uncertainty in all its movements; it refuses food, the respiration becomes irregular and slow, and death takes place in from four to eight hours.

In such a case the greater part of the injected fluid is found in the subcutaneous cellular tissue of the back in much the same condition as before it was injected. It contains bacteria of the most diverse forms, irregularly mixed together, and as numerous as when examined before injection. No inflammation can be observed in the neighbourhood of the place of injection. The internal organs are also unaltered. If blood from the right auricle be introduced into another mouse, no effect is produced. Bacteria cannot be found in any of the internal organs, nor in the blood of the heart.

Koch therefore concludes that an infective disease has not been produced as the result of the inoculation, but that the death of the animal is due to a soluble poison—Sepsin—which has been shown by the researches of Bergmann, Panum, and others to exist in putrid blood. This supposition is confirmed by the fact that when less fluid is injected the symptoms of poisoning which follow are less marked, and are quite absent when one or at most two drops have been injected. After the use of such small quantities mice often remain quite well, but

a third of them, on an average, become ill after the lapse of about twenty-four hours. The less amount of **septic** fluid injected, the fewer mice become affected, but less than one drop is sometimes sufficient; thus, of twelve inoculated with one-twentieth to one-tenth of a drop each, only one was successfully infected.

The first symptoms of the success of the inoculation with this minute quantity is an increased secretion from the conjunctiva. The eyes appear dull, and a whitish mucus collects between the lids, and finally glues them together. At the same time lassitude sets in, the animal moves little and languidly; as a rule it sits quite still, with its back much bent and its extremities closely drawn up. It then ceases to eat; its respirations become slower; weakness gradually increases, and death comes on almost imperceptibly. Convulsions never precede it (they always do so in anthrax). After death the animal still remains in the sitting posture with its back strongly bent; whilst a mouse which dies from anthrax is always lying on its back or side, with its stiffened limbs fully extended. Thus, by the position of the body after death, a fatal result produced by the inoculation of putrefying blood is at once distinguished from that occasioned by inoculation with the material of anthrax.

On *post mortem* examination there is found at the place of injection or inoculation slight œdema of the subcutaneous cellular tissue. This, however, is often absent, and the internal organs, with the exception of a considerable swelling of the spleen, appear quite unaltered. One-tenth of a drop of the fluid of the subcutaneous œdema, or of blood from the heart of such animal, inoculated into another mouse, is sufficient to cause exactly the same symptoms, and death in about fifty hours. From this second animal a third may be injected, and so on through as many successive animals as one pleases.

Koch says that the certainty with which the infective material can be carried from one mouse to another is greater than in anthrax, as in the latter the material for inoculation must be taken from the spleen, because the blood of mice affected with anthrax often contains very few bacilli; but in septicæmia, produced by putrefying blood, it is a matter of indifference from which organ the material for inoculation is taken, and even the smallest quantity will produce an effect with certainty.

The blood of mice which became ill after intravenous *injec-*

tion of one to ten drops of putrefying blood was found to contain, as a rule, different varieties of bacteria in small numbers, micrococci, and large and small bacilli. If, however, the animals died after *inoculation* with putrefying or septicæmic blood, small bacilli alone appeared. This result was invariable, and the bacilli were in large numbers.

These bacilli are often attached to each other in septicæmic blood in pairs, either in straight lines or forming an obtuse angle. Chains of three or four bacilli also occur, but they are rare. They show at first sight a great resemblance to small needle-like crystals, but that they are undoubtedly vegetable bodies is evident, for when septicæmic blood is kept in an incubation apparatus, these grow in a manner similar to the bacilli of anthrax, not forming long threads like the latter, but dense masses, which consist of isolated bacilli sometimes containing spores. They are extremely difficult to see, and it is necessary to stain them.

The relation of these bacilli to white corpuscles of the blood is peculiar. They penetrate into these and multiply in their interior, and it is often found that there is hardly a single white corpuscle in the interior of which bacilli cannot be seen.

Disease induced by the inoculation of the blood of septicæmic mice cannot so successfully be induced in so many different animals as with that of anthrax. Thus Koch failed to cause it by inoculating rabbits, and even field mice resisted the inoculations.

Occurring along with the septicæmic bacillus, Koch has found in mice, after the introduction of putrefying blood, a micrococcus in the neighbourhood of the place of injection. This organism is of rapid growth, and forms very regular chains. As a rule, when an animal dies of septicæmia after about two days, none of the numerous forms of bacteria which were injected with the putrid blood can be discovered, except the septicæmic bacilli, or it may be a few residual specimens growing with difficulty. It must therefore be supposed that none of the other bacteria injected at the same time find in the body of the living mouse a suitable soil, and that they therefore die more or less quickly. Koch's attention was therefore arrested when he found in some cases, growing at and about the inoculated spot, micrococci in unusual abundance and of a constantly characteristic form.

They were not present in the blood, and by inoculation with the blood the septicæmic bacilli alone were transmitted. In order to test whether they could be inoculated, it was necessary that the material should be taken from the place of injection. Inoculations carried out in this way were successful in producing both forms of disease, and the virulence of the serum from the subcutaneous cellular tissue containing these micrococci was just as marked as that of the septicæmic blood. When the point of a knife which had been well cleaned was merely brought in contact with the subcutaneous tissue at a spot about one centimetre and a half from the place of injection or inoculation, and when with this knife another animal was immediately inoculated, the inoculation was successful on every occasion. Septicæmia was of course always produced at the same time, because the serum used contained also septicæmic bacilli. The influence of these micrococci on animal tissues, and their mode of spreading, can be best traced on the ear of a mouse, and it is specially instructive to compare an ear on which only septicæmic bacilli have been inoculated with one into which both the bacilli and the chain-like micrococci have been introduced. In the former the cellular tissue is full of red corpuscles and lymph cells, so that the bacilli can often be recognised only with great difficulty among the numerous cell nuclei. The other ear presents totally different appearances. Spreading from the place of inoculation, one can see extremely delicate and regular micrococcus chains, here pressed together so as to form thick masses, traceable almost to the base of the ear, all the tissues of the part occupied by them being remarkably altered. As far as the micrococci extend, neither red blood globules nor nuclei of lymph or of connective tissue cells can be seen. Even the extremely resistant cartilage cells, and the plasma cells so richly present in the mouse's ear, and which are likewise characterised by great resisting power, are pale and scarcely recognisable. All the constituents of the tissue look as if they had been treated with caustic potash; they are dead, they have become gangrenous. Under these circumstances the bacteria develop all the more vigorously, and the micrococci penetrate in numbers into the damaged blood and lymphatic vessels, and here and there they fill them so com-

pletely that the vessels appear as if injected. Among these the septicæmic bacilli, no longer obscured by nuclei, are seen very distinctly in small groups, which are at times very dense, and can be traced up to the root of the ear, and indeed beyond it, having at the same time increased enormously in the blood, and ultimately causing the death of the animals. The micrococci, on the other hand, and the destructive process associated with them, have only extended during the same time (about fifty hours) as far as the vicinity of the root of the ear, where their limit is sharply defined. The appearances presented by the ear on a *post mortem* examination lead to the conclusion that the action of these micrococci in causing gangrene is somewhat as follows:—Introduced by inoculation into living animal tissues, they multiply, and as a part of their vegetative process they excrete soluble substances, which get into the surrounding tissues by diffusion. When greatly concentrated, as in the neighbourhood of the micrococci, this product of the organisms has such a deleterious action on the tissue cells that they perish and finally completely disappear. At a greater distance from the micrococci the poison becomes more diluted and acts less deleteriously, only producing inflammation and accumulation of leucocytes. Thus it happens that the micrococci are always found in the gangrenous tissue, and that in extending they are preceded by a wall of nuclei, which constantly melts down on the side directed towards them, while on the opposite side it is as constantly renewed by deposition of fresh leucocytes.

These observations refer to inoculations with fluid containing both micrococci and bacteria, and it might have been supposed that the septicæmic bacteria were necessary forerunners of the micrococci, and must to a certain extent prepare the way for them.

Koch attempted by various means to separate them from each other, but for a long time with no avail. But chance led him to the proper method. A field mouse, which he had already found out possessed an immunity from septicæmia, was inoculated with bacteria and chain-like micrococci. The experiment was made in the expectation that neither parasite would develop. This expectation, however, was not fulfilled, for, though the rods, as usual, underwent no development, the

micrococci increased and spread in exactly the same manner as had been the case with the domestic mouse.

Beginning at the point of inoculation in the root of the tail, the gangrene spread onwards along the back, passing deeply among the dorsal muscles, and downward on both sides to the abdominal wall. The animal died three days after inoculation. The parts affected with the gangrene were partially denuded of epidermis and hair, and contained chain-like micrococci in extraordinary numbers. The same micrococci were also found on the surface of the abdominal organs, although there was no visible peritonitis. The blood and the interior of the organs were, on the other hand, quite free from them. From this animal other field mice, and from these again domestic mice in various successive series, were subsequently injected, and with always the like result—namely, that only chain-like micrococci, and in their train progressive gangrene, were obtained.

The development of bacteria even in a suitable soil can be checked by certain influences, physical, biological, and chemical, and of these none are more potent than temperature. No fully developed bacteria have as yet been found to survive after long exposure to 60° C. when in the moist state, and but few resist a temperature of 50 °C. = 112° F.

The spores of bacteria, however, can resist a much higher temperature than that which destroys the adult organism, and all agree that the boiling-point of water is the lowest which can be relied upon to destroy spores; but in the dried state they are much less readily destroyed, requiring three hours' exposure to a temperature of 140° C. = 284° F.

Again, intense motion and sunlight checks bacterial life, but electricity seems to have no effect upon them.

Of the chemical influences which destroy or retard bacterial life the most powerful is corrosive sublimate, its action being instantaneous even in a dilution of one part to five thousand. Next to the bichloride are placed bromine, iodine, and chlorine; but carbolic acid is not so reliable, as nothing less than a three per cent. solution destroys spores, and requiring at least seven days, and a five per cent. solution requiring from twenty-four to forty-eight hours. When dissolved in oil, Koch says it is harmless both to spores and developed bacteria. When immersed in it in the dried state and dissolved in alcohol, it is also inert.

## CHAPTER XI.

### PATHOLOGY—*continued.*

#### MODES OF DEATH.

FOLLOWING the example of Dr. Williams, Sir Thomas Watson, and other writers upon medicine, I purpose very briefly to consider the chief varieties of the modes of death.

An inquiry into this subject, says Watson, “is not one of merely curious interest, but has a direct bearing upon the proper treatment of disease. It will teach us what we have to guard against, what we must strive to avert in different cases.”

A continuance of “life is inseparably connected with the continued circulation of the blood. So long as the circulation goes on, life, organic life at least, remains. When the blood no longer circulates, life is presently extinct; and our investigation of the different modes of dying resolves itself into an investigation of the different ways in which the circulation of the blood may be brought permanently to a stand.

“Observe the ample provision that is made in the construction of the body for carrying on and maintaining this essential function. First, there is an extensive hydraulic apparatus distributed throughout the frame, consisting of the heart and other blood-vessels. Next, there is a large pneumatic machine, forming a considerable part of the whole body, composed of the lungs and the case in which they are lodged. Lastly, the power by which this machine is to be worked and regulated is vested in the nervous system. Each of these systems must continue in action, or the circulation will stop, and life will cease. The functions they respectively perform are consequently called vital functions; and their main organs—the heart, the lungs, the brain—are denominated vital organs.

The functions of any one of the three being arrested, the functions of the other two are also speedily extinguished.”—  
(WATSON'S *Principles and Practice of Physic.*)

Death, it is observed, occurs as follows :—

1. Death beginning at the heart—syncope.
2.    „            „            „    lungs—apnoea.
3.    „            „            „    brain—coma.
4.    „            „            „    blood—necræmia.

#### DEATH BEGINNING AT THE HEART.

Death beginning at the heart may occur in two ways :—  
1st. Suddenly, or by syncope ; and 2nd. By gradual cessation of the heart's action. Syncope may occur in two ways :—  
1st. By the heart losing its irritability, so that it ceases to contract ; and 2nd. By tonic spasm, in which it remains rigidly contracted. Death from tonic spasm of the heart is rare amongst the lower animals. I have, however, seen one instance, where a valuable horse died from this cause in consequence of a sudden fright.

In both these cases death is instantaneous. In the first case, each chamber of the heart is found after death to be filled with its proper kind of blood, upon which it has been unable to contract. Sir Benjamin Brodie, in some experiments recorded in the *Philosophical Transactions* for 1811 and 1812, found that when death was occasioned by the *upas antiar*, that the heart was full after death, with purple blood in its right and scarlet blood in its left cavities. This proves that the heart has ceased to contract upon the contained blood. In the second case, the heart appears very small and hard, the ventricles, particularly the left, firmly contracted and containing no blood, and the heart substance very firm. This state of the heart was supposed to depend upon concentric hypertrophy, but its true nature was pointed out by Cruveilhier and Dr. G. Budd. Should any doubt remain as to its true nature, if the heart be kept for a few days, it will, if the condition be that of spasm, lose this tonicity, and may easily be restored to its regular dimension by a little manipulation. Both conditions of the heart—namely, loss of irritability and tonic spasm—may arise from similar causes. *Shock*, for example, may produce the one or the other



condition; and wounds of the heart are sometimes followed by paralysis, sometimes by rigid contraction of that organ. In sudden death from drinking very cold water when the body is heated, the heart has been found contracted.

In the slower modes of death beginning at the heart, it is found that the same conditions exist after death. In death from hæmorrhage—anaemia—it is found that the heart is firmly contracted, if the animal be examined shortly afterwards; the circulation failing, not because the heart has lost its power of contraction, but because blood does not arrive in sufficient quantities in its cavities. That the heart does not lose its power of contraction in such cases is proved by the transfusion of blood from an animal into the veins of a human being or other animal apparently dead from hæmorrhage, where it has been found that animation may thus be restored. "It is quite clear," says Watson, "that this introduction of fresh blood could be of no avail in a case where the heart was unable to act upon the blood which had already reached it."

Approaching death from hæmorrhage is manifested by great and rapidly increasing pallor of the mucous membranes, coldness of the mouth, surface of the body, and extremities, cold sweats, a dazed or dim appearance of the eyes, convulsive struggles—if the animal is down, at first strong but rapidly becoming feeble, if standing, the animal will balance its body upon the extremities; the pulse feeble, irregular—sometimes slow, sometimes fast—cold breath, convulsive breathing, dilatation of the pupils, and very often just before death the animal gives a loud scream or delirious neigh.

In another form of gradual death beginning at the heart, it is found that the heart has gradually lost its power of contractility. This is the most common mode by which death occurs, and is the termination of many exhausting diseases, especially those which destroy the strength without directly interfering with the more vital functions, such as influenza, diarrhoea, diabetes, fevers, as well as inanition from want of food, or other causes which reduce the powers of the whole body.

The two conditions of the heart found after death beginning at that organ may each be brought about in two ways:—*1st.* Suddenly, as in instantaneous death from shock or great violence; and *2d.* When death, although it may be said to occur suddenly,

presents some symptoms of its approach, however brief those symptoms may be, as from hæmorrhage, the operation of some poisons, as aconite, hydrocyanic acid, upas antiar, tobacco; from lightning, electricity, and various animal poisons, as snake-bites; the operation of debilitating influences, as well as diseases which tend to weaken and paralyze muscular force, and to produce asthenia.

In death from gradual asthenia the pulse becomes very feeble, frequent, and often irregular; the respirations feeble, sometimes sighing; the muscular debility extreme. Pallor of the membranes comes on gradually, or in some cases they may present an unhealthy reddened or brownish-red appearance from the blood remaining in the veins, the extremities become cold, and œdematous.

In various wasting diseases, such as pleuro-pneumonia-bovina, diarrhœa, polyuria, or any disease which more gradually exhausts the powers of life, death is caused by anæmia and asthenia. The blood gradually diminishes in quantity and deteriorates in quality, therefore the supply to the substance of the heart becomes diminished, and in a condition unsuited to nourish the heart properly. The power of the heart, as well as of the muscles generally, is gradually lessened, and at length ceases altogether; and when death is so produced the heart is not contracted and empty as in death from anæmia, nor so full and engorged as in death from asthenia. This form of death is called by Watson *death by inanition*, and its typical form is that occurring from starvation.

#### DEATH BEGINNING AT THE LUNGS.

By this it is meant that the function of breathing is the first to fail, improperly termed death from asphyxia—literally pulselessness—more correctly termed death by apnœa, privation of breath, or suffocation.

This form of death is caused by diseases of the lungs or bronchial tubes, pleurisy terminating in hydrothorax, where air is prevented from entering the lungs by the pressure of the pleuritic fluid, mucus, pus, or blood, filling the tubes and air cells; by laryngitis, spasm of the glottis, or when tumours press upon the larynx or trachea; by œdema of the glottis; diseases of the

heart affecting the quantity of blood in the lungs and great thoracic vessels; strangulation; drowning; pressure upon the diaphragm, forcing it forward and preventing admission of air into the lungs, as in severe tympanitis of the stomach and bowels, or from any circumstance which may paralyze the diaphragm—fracture or dislocation of the upper cervical vertebrae, and pithing, for example—or obstruct the passage of air through the nostrils, larynx, trachea, or bronchial tubes.

Sudden death from apnoea is not often witnessed as the result of disease, but is generally brought about by accident or design, accidentally when the upper cervical vertebrae are fractured or displaced; in cases of parturient apoplexy, when fluids gain entrance into the lungs, and from impaction of foreign bodies, or polypi in the larynx; and designedly, when an animal is pithed. Death is also thus produced by the entrance of air into the veins.—(See *Principles and Practice of Veterinary Surgery*.)

Death from apnoea takes place most commonly from the supply of air being gradually cut off by morbid changes in the respiratory organs, and is often accompanied by asthenia and coma; but generally the symptoms belonging to apnoea are plainly predominant. In death, when the passage of air into the lungs is arrested suddenly and completely, it has been observed that the muscles of respiration exhibit strong and violent contractions; that the efforts to breathe are very great, struggling but ineffectual, and very distressing. This extreme listress, however, soon passes away, and is succeeded by vertigo, stupor, loss of consciousness, and convulsions, till at length all efforts cease, except a few irregular twitchings of the limbs; the muscles then relax, the sphincters yield, but even then the movements of the heart and the pulse continue for a short time after all signs of life are gone. The other signs of this method of death are congestion and lividity of the visible mucous membranes, a full, staring eye, protrusion of the nose, dilatation of the nostrils, and sometimes flapping of them. When it arises from inflammation or spasm of the muscles of the larynx, tumours, abscesses, or obstruction in the trachea, there will be a loud, roaring, inspiratory sound. In the slower forms of apnoea, from diseases of the lungs, air tubes, or hydrothorax, where the interruption is less complete, the efforts less violent, the congestion of the membranes less marked, there may be little

perceptible protrusion of the nose ; indeed, in bronchitis, stupor is present to such a degree that the animal generally hangs its head. The functions of the body together gradually failing, the symptoms of suffocation are less decided.

The pathology of this mode of dying is now pretty well understood. Bichat made several experiments which went to prove that unaërated blood not only reached the left side of the heart, but was sent to all the arteries of the body.

His experiments were as follows:—A ligature was applied to the trachea of a living animal ; a small opening was then made in one of the carotid arteries. Presently the slender stream of blood which issued began to lose its arterial tint, and to assume the dark colour of venous blood ; but, contrary to what had been previously supposed—namely, that the circulation would be immediately arrested in the lungs, the quiescence of the lungs, consequent upon the cessation of the alternate movements of the thorax, forming a mechanical impediment to the transit of blood through them (Haller), or that the unaërated blood passed through the lungs and entered the left auricle and ventricle, but went no farther (Dr. Godwin)—Bichat discovered that the blood continued to flow from the opened carotid, and that its afflux upon the brain was marked by convulsions and insensibility. This led him to believe that the blood underwent *no* obstruction in its passage through the lungs, but that, remaining unpurified and venous, it acted as a poison upon every part to which it was carried by the arteries—first upon the nervous system, and ultimately (passing through the coronary arteries) upon the muscular substance of the heart itself. “There are, however,” says Watson, “two well-known facts upon which this theory is inexplicable—the comparative emptiness of the left chambers of the heart, and the restoration of the suspended functions by the timely performance of artificial respiration. The air could never reach and revivify or depurate the venous blood stagnating in the capillaries of the heart.” Sir James Kay Shuttleworth, in his work on Asphyxia, and later, Mr. Erichsen, in the *Edinburgh Medical and Surgical Journal*, have endeavoured to prove that stagnation of the blood commences in the pulmonary capillaries, that the stagnation is due to its non-arterialization, and that the movements of the left side of the heart are brought to an end principally by the deficient supply of blood from the lungs.

Venous blood (say these observers), circulating through the arteries, has no directly poisonous operation, but is capable, though much less effectually than arterial blood, of supporting in some degree the irritability of the muscles; and it appears that the primary and main cause of the failure of the circulation seems to be the difficulty with which the non-arterialized blood passes through the capillaries of the lungs, and partly the imperfect stimulus afforded by the venous blood to the walls of the heart, as well as its incapability to support the functions of the brain and nervous system. The appearance of engorgement of the pulmonary arteries and venous system generally, with distension of the right side and comparative emptiness of the left side of the heart, seen after death, are more constantly visible in the slower forms of apnoea than after sudden suffocation; for after sudden death blood seldom coagulates, and if the animal is not shortly examined, the engorgement is apt to disappear by gravitation.

The conclusions of Sir James Kay Shuttleworth and of Mr. Erichsen, although correct enough, fall rather short of a full explanation; for it has been ascertained by many observers that when an animal is strangled by a tight ligature on the trachea, and examined immediately after death, the lung tissue is always found void of blood, whilst the pulmonary artery, up to its smallest branches, the great veins and the heart, are greatly engorged. Dr. George Johnson, in explaining this, says that some opposing power is brought into play, more than equal to the propelling power of the heart, and that this opposition is due to the firm contraction of the muscular walls of the ultimate branches of the pulmonary artery. He calls this contraction the *stop-cock* action of the ultimate pulmonary arteries. Dr. Johnson proved by experiments of his own that this condition exists whether the ligature be applied after or before a full inspiration; whether the lungs were at the time full or comparatively empty of air. "These," says Watson, "are the plain and unquestionable facts of the case. They show that some opposing power must have been called into play more than equal to the propelling power of the right ventricle of the heart. Now, such a power—and it is the only conceivable one—actually exists at the very place where the venous current meets with its curb, and it consists in the firm contraction of those muscular

fibres of the minute arteries, the function of which is to regulate the blood supply in accordance with the varying requirements of the part. This function again is determined by those unsleeping sentinels the (vasa-motor) nerves. Were it allowable, for the sake of illustration, to impersonate the vital forces concerned in this marvellous adaptation, we might liken the process to the intelligent stopping of the traffic on an obstructed line of railway by a backward telegram."

In order to test the correctness of Dr. George Johnson's experiments, I have repeatedly examined strangled dogs, and found that the heart will continue to beat for more than three minutes after the respiratory efforts have ceased, and that the lungs and great vessels are always as he has described them, namely, the lung substances pale and empty, the pulmonary artery and right side of the heart, with the cavæ, immensely engorged, and the pulmonary veins and left side of the heart containing a small quantity of *venous* blood. From these experiments it appears very conclusive that death by apnoea occurs in two ways, and that two distinct pathological conditions of the lungs are observable after death:—*1st*. When death is sudden, as from suffocation by strangulation, drowning, or any cause whereby the supply of air is suddenly cut off, the stop-cock action of the ultimate pulmonary arteries prevents the blood from flowing in its ordinary quantity to the pulmonary capillaries, veins, and left side of heart, and that the lungs are pale when examined immediately after death: *2nd*. When death more slowly occurs, the blood accumulates in all the vessels of the lungs, which after death appear engorged, congested, and black. This is the condition of the lungs seen when an animal is galloped to death, in pulmonary apoplexy, and congestion; and in death from some acute diseases, where towards the close of the disease the heart becomes too enfeebled to propel the blood through the intricacies of the pulmonary system, or when the blood itself becomes so altered as to be incapable of proper arterialization.

To prevent death by apnoea, it is obvious that the respiratory function must be restored where it is defective, that all impediments to the passage of pure air into the lungs must be removed. The subject of treatment will, however, be considered in another place, and I may merely state here that nothing has been found

so effective and so useful in balancing the circulation and in restoring the passage of blood through the lungs as the application of heat to the whole body, frictions, stimulating applications to the extremities, warm clothing, and bandages, in addition to abundance of pure air for the animal to breathe, and that blood-letting frequently affords apparent relief.

#### DEATH BEGINNING AT THE BRAIN.

Death by coma, or beginning at the brain, is much less commonly witnessed in the lower animals than death by apnoea or by asthenia. Such diseases, however, as parturient and idiopathic apoplexy, cerebral meningitis, and other allied affections, which act upon and destroy the functions of the brain, cause death in this way. Death by coma is also induced by certain narcotic poisons, such as opium, as well as by inordinate quantities of effete materials in the blood, more especially urea and carbonic acid; by fractures of the cranial walls, the pressure of tumours, abscesses, serum or extravasated blood, and by coagula in the cerebral arteries, obstructing the flow of blood, and causing anæmia of the cerebral mass.

The symptoms of coma are stupor, insensibility, suspension of voluntary motion, which come on sometimes suddenly, as in apoplexy and injuries of the head, whilst in other cases they supervene gradually. The breathing becomes slow, irregular, stertorous; the instinctive motion of breathing still continues, but all voluntary attention to the act is lost. The feeling of the want of air is still sufficiently strong and powerful to excite, through the medium of the pneumo-gastric and branches of the fifth nerves, the reflex power of the medulla oblongata to sustain the involuntary movements of the thorax; but at length this fails also, the chest ceases to expand, the blood is no longer aerated, and thenceforward precisely the same internal changes occur as in death by apnoea.—(WATSON.)

Coma ultimately destroys life in the same way as apnoea, with this difference, that in death by apnoea the aëration of the blood and the functions of the lungs cease first—the circulation of the non-arterialized blood destroying the functions of the brain; whilst in coma the functions of the brain cease first, and in consequence of the loss of nervous power, the movements of

the chest and the aëration of the blood cease also. The *post mortem* appearances of death by coma and of that by apnoea are the same, except, indeed, in those cases where the cause of the coma remains, when it will be present in addition to those of apnoea. Reasoning upon the conclusion that the circulation ceases consequent upon the cessation of the act of respiration, arising from suspension of the nervous power, Sir Benjamin Brodie was led to think that by continuing respiration artificially in animals labouring under narcotic poisons, the circulation of the blood might be kept up until the effect of the poison upon the brain had passed off. The correctness of the supposition he soon verified by experiment. He inserted some woorara into a wound which he had made in a young cat: this after a short time entirely destroyed the respiratory movements, and the animal appeared to be dead, but the heart could still be felt beating. The lungs were then artificially inflated about forty times a minute. The heart continued to beat regularly. When artificial respiration had been kept up for forty minutes, the pupils of the cat's eyes were observed to contract and dilate upon the increase and diminution of light, but the animal remained perfectly motionless and insensible. At the end of an hour and forty minutes there were slight involuntary contractions of the muscles, and every now and then there was an effort to breathe. At the end of another hour the animal, for the first time, showed some signs of sensibility when roused, and made spontaneous efforts to breathe, twenty-two times in a minute. The artificial breathing was, therefore, now discontinued. It lay as in a state of profound sleep for forty minutes longer, when it suddenly awoke and began to walk about. It must be clearly understood that artificial respiration can be beneficial only where there is a *suspension* of the nervous functions, as in cases of poisoning, and not where there is destruction of them by disease, injury, or the long-continued action of poisons.

Professor Sewell experimented upon the horse with the woorara poison, and found that by artificial respiration life could be maintained until the action of the poison had passed away, and that afterwards the animal regained consciousness and recovered from its effects.



## DEATH BEGINNING WITH THE BLOOD.

*Necræmia, or death beginning with the blood.*—In rinderpest, splenic apoplexy, black-quarter or quarter-ill, braxy, purpura hæmorrhagica, and scarlatina, death may be said to be due to the death and decomposition of the blood, the action of the heart ceasing because the blood is no longer capable of affording the necessary stimulus.

At an early stage of these diseases, when they occur in their worst form, the blood exhibits changes in its composition, manifested by petechiæ and vibices on the skin and mucous membranes, extravasations into the subcutaneous and muscular tissues, and by congestion of, and hæmorrhages into, the internal organs.

The blood is fluid, of a dark colour, and possesses pathogenic properties, as manifested in its deleterious operations on other animals and on man.—(See *Malignant Pustule*.) It decomposes rapidly, and a putrid odour is exhaled from the surface of the body, and from the excretions.

“The blood,” says Dr. Williams, “the natural source of life to the whole body, is itself dead, and spreads death instead of life. Almost simultaneously the heart loses its power; the pulse becomes weak, frequent, and unsteady; the vessels lose their tone, especially the capillaries of the most vascular organs, and congestions occur to a great amount; the brain becomes inactive, and stupor ensues; the medulla is torpid, and the powers of respiration and excretion are imperfect; voluntary motion is almost suspended; secretions fail; molecular nutrition ceases; and, at a rate much more early than in other modes of death, *molecular* death follows close on *somatic* death—that is, structures die, and begin to run into decomposition as soon as the pulse has ceased; nay, a partial change of this kind may even precede the death of the whole body (*Somatic Death*—Dr. PRITCHARD); and the foetid aphthous patches in the throat, the offensive colliquative diarrhœa of persons in the last stage of various fatal diseases, parts running into gangrene, as in carbuncle, the sphacelous throat of malignant scarlatina, and the sloughy sores of the worst forms of typhus, and in the large intestines in dysentery, and the putrid odour exhaled even before death by the bodies of those

who are the victims of similar pestilential diseases—are so many proofs of the early triumphs of dead over vital chemistry.”—WILLIAMS' *Principles of Medicine*.)

This mode of death is not recognised by some writers, but the truth of the above quotation will, I have no doubt, strike many who have witnessed the course and progress of the blood diseases of the domesticated animals; the extensive congestions and extravasations of black, tar-like blood, and its rapid decomposition in splenic apoplexy and quarter-ill; the gangrenous throat, and sphacelated patches of skin in purpura hæmorrhagica and scarlatina; the putrid odour of the secretions and the aphthous patches of rinderpest, and the very general advent of an exhaustive diarrhœa in those cases which do not succumb to the shock of the disease; the peculiar odour exhaled, as well as the rapid decomposition of the whole body after death;—indeed it may truly be said that decomposition of the blood has set in prior to death in many instances.

The symptoms of the approach of death by necræmia are generally as follows:—A dusky livid colour of the visible mucous membranes; slight exanthematous patches on the skin, petechial spots—in rinderpest these were thought to be small-pox pustules—or extensive extravasations and the production of a foetid gas subcutaneously; oozing of a thin sanguineous fluid from the nostrils or intestinal canal, or of blood in a semi-coagulated condition, extreme prostration of strength; the pulse accelerated, feeble, soft, sometimes presenting a double beat; twitchings of the limbs; dilatations of the pupils, the eye often fixed and staring, but sometimes with the lids closed; unequal and frequent respirations; cold perspiration, and a gradual fall of the animal temperature. The horse will generally stand until the last, and will only fall from prostration; it will then struggle ineffectually to rise. The cow and sheep, however, will lie down in the earlier stages of disease, and scarcely ever struggle to rise.

The prognosis in all diseases which tend to terminate in necræmia is seldom or never favourable, but it is generally considered expedient to endeavour to avert the tendency to a fatal termination; and there are certain considerations which may be useful guides to the practitioner to effect this end—namely, 1st. That such diseases tend to cause congestions of some organ or organs, as the lungs, spleen, stomach, and bowels, and conse-

quently means should be taken to prevent as much as possible the occurrence of such congestions by removal of the patient from the contamination of foul air or bad drainage, and the avoidance of gastric or intestinal irritants. 2*d*. That the blood to a varying extent loses its plasticity, and is apt to exude through the walls of the vessels into the looser and softer tissues, and there undergoing rapid decomposition, causes gangrene of the tissue with which it is in contact, as in black-quarter, &c.

Means must therefore be taken to overcome this condition of the blood-mass by the administration of the chlorate of potash, which has a remarkable effect in restoring both colour and plasticity to the altered blood by increasing its coagulability; oil of turpentine, which is not only a most valuable antiseptic, but is also a styptic, and in virtue of its diuretic properties assists the kidneys in the removal of effete materials. Quinine, the mineral acids, and the salts of iron will be useful at a later stage, should convalescence be established. Ammonia and its salts are to be avoided, as they tend to decrease the blood's coagulability. Eggs, milk, and, as some recommend, beef tea, may, however, be administered in the earlier stages of the maladies, and continued until there be a return of appetite.

## CHAPTER XII.

### PATHOLOGY—*continued*.

#### CLASSIFICATION OF DISEASES.

A correct classification of diseases, at once simple and suggestive, is a subject of profound importance, essential to the existence of veterinary science, and necessary for its teaching; and were the veterinarians of Europe to assemble in a great congress, and there agree upon a system by which diseases might be named, defined, enumerated, and classified, they would indeed attain a great object. At the present time, however, the science of *nosology* is very unsatisfactory as applied to the diseases of the domesticated animals, and incomplete even in the science of human medicine.

In a work of this kind it is necessary that I should refer to the efforts of physicians to arrive at a satisfactory system of nosology, and by reading books on medicine it will be found that diseases have been classified in different ways. In the system of Sauvages diseases were divided into ten classes—*vitia, febres, phlegmasiæ, spasmi, anhelationes, debilitates, dolores, vesaniæ, fluxus, cachexia*. Linnæus, Vogel, and Sagar's classifications were also of this kind, and Cullen's method was an unnatural simplification of it, by which all diseases were arranged in four classes—*pyrexia, neuroses, cachexia, and locales*. This system of classification is based upon the symptoms, and regards them as the essences of diseases; hence it is exceedingly unsatisfactory, and has been abandoned.

Believing that nosology should be founded upon correct pathology, Pinel divided diseases into five kinds, namely, *fevers, inflammations, hæmorrhages, neuroses, and organic affections*. This classification is necessarily imperfect from the state of pathological science in Pinel's days, but it approaches very

nearly the standard of a pathological nosology; was further developed by Bichat, and adopted by Dr. Craigie in 1836.

The results of modern investigations into disease prove that many ailments hitherto supposed to be merely functional are in reality accompanied by organic alteration of tissue; and it is not unreasonable to suppose that many of the so-called functional diseases will be found to depend upon an alteration of structure, and even where no organic change can be detected in cases where the existence of disease cannot be doubted, we may attribute our failure to the imperfection of our means and instruments of investigation, or our modes of using them.

Dr. Aitken, to whose works the reader is referred for further information, observes that "a perfectly philosophical or natural system of classification aims at having the details of its plan to agree in every respect with all the facts as they exist in nature, and to be, as it were, a 'translation of the thoughts of the Creator into the language of man.' To effect this end, arrangements, as they *naturally* exist, require to be traced out, not devised. The tracks in which such a pursuit must be followed up, and in which our knowledge is as yet deficient, may be shortly indicated under the following heads, namely:—

"(1.) The affinities or alliances of diseases with each other. (2.) The morbid anatomy of diseased parts. (3.) The communication, propagation, inoculation, generation, development, cause, and spontaneous natural termination of diseases. (4.) The connection of the phenomena recognised during life with the facts of morbid anatomy. (5.) The geographical distribution of diseases. (6.) The succession of diseases, as far as they can be traced through past ages; the peculiarities they have exhibited at different periods in the world's history, or within comparatively recent cycles of years.

"But the time has not yet come for a classification on a basis so comprehensive, simply because the material does not exist; and attempts to make so-called *natural* systems of arrangement must end in disappointment, on account of the uncertain and fluctuating data on which they must be based."

The arrangement of animal diseases, and their division into classes, groups, and orders, is a very difficult matter. They might certainly be arranged according to their pathology, to their causes, to the tissue or the systems of the animal body

affected, or anatomically, according to the parts which they invade. The simplest plan, however, seems to me to be that adopted by Reynolds, namely, the division of diseases into two great groups.

The first group includes those diseases in which the whole organism appears primarily and prominently deranged, and the second group those diseases in which special organs or systems of organs are in like manner affected. The first group is divided into two classes:—A, those in which the disease appears to be developed by causes operating from without; and B, those in which it depends upon change within the body. In the first class we have those diseases which are caused by atmospheric influences, contagion, and other external causes; and in the second, hereditary diseases, rheumatism, rickets, and other obscure bone diseases.

The second group, or that in which special organs are diseased, is again subdivided into many classes, consisting of diseases of systems of organs, such as—A, diseases of the nervous system; B, diseases of the digestive system and its appendages; C, diseases of the circulatory system; D, diseases of the respiratory system; E, diseases of the urinary system; F, diseases of the reproductive system; G, diseases of the locomotive system; and H, diseases of the cutaneous system.

## CHAPTER XIII.

### CONTAGION AND CONTAGIOUS DISEASES.

THE origin of contagious diseases has been, and is yet to some extent, a matter of controversy. It is, however, generally accepted that they are caused by specific pathogenic micro-organisms, having the power of inducing specific morbid lesions when introduced into the animal body.

Many writers assert that a contagious disease can only arise from the introduction of a specific microbe existing in the fluids and solids of a diseased animal into the body of another, and Koch laid down the following postulates, to which it was supposed there could be no exceptions; but further observations have proved that there are many exceptions to his conclusions, and an explanation is thus given to the belief in a spontaneous origin of a contagious disease. Koch's postulates are as follows:—

1. The micro-organism must be found in the blood or diseased tissue of men or animals suffering from or dead of the disease.

2. These micro-organisms must be isolated from the blood, lymph, or tissues, and cultivated in suitable media outside the animal body. To obtain these cultivations pure they must be carried on through successive generations of the organism.

3. A pure cultivation thus obtained must, when introduced into the body of a healthy animal, produce the disease in question.

4. Lastly, in the inoculated animal the same micro-organism must again be found.

Although coming from such a high authority, these postulates must not be accepted in their entirety, for we have many microbic diseases where this form of origin cannot possibly have an

existence. For example, septicæmia originates from the accidental introduction into the circulation of surrounding external micro-organisms, naturally existing in the air, or within the body itself. Again, tetanus, now recognised as a microbic disease, is induced by an organism located in the ground. These examples might be multiplied, but will be referred to when dealing with microbic diseases specially. It may, however, be mentioned that these so-called non-pathogenic microbes are classified under the term saprogenic or putrefactive bacteria, and acknowledged that they play an important part in the economy of nature. The Koch postulates may be added to by including such diseases as rabies, rinderpest, South African horse-sickness, the poxes, &c., all of which are due to an organism, which is, however, too small to be seen with the present microscopes. We might also include those diseases which are due to an organism which has not yet been cultivated artificially, such as the *Cryptococcus farcinosus* of epizootic lymphangitis, and the protozoan organisms, such as trypanosomata and piroplasmata.

Some of these multiply in dead organic matter, others proliferate accidentally within the body, their natural habitat being the external surroundings; but many of them can lead a double existence, saprophytes growing on dead organic matter, and parasites which lead their existence in a living host.

Parasites which exist in and on living matter only are termed *obligatory parasites*; those that lead a double existence *facultative parasites*; and in addition to these there is another class, called *non-contagious facultative parasites*, which live in their normal condition in the external media, and only incidentally develop within the living organism—which development is due to some loss of resistance on the part of the animal attacked. For instance, the *Bacillus coli* is a normal inhabitant of the intestine, but if an animal becomes low in resistance, due to the attacks of animal parasites (worms) or other cause, this organism may enter the liver and form an abscess, or set up an enteritis of the large intestine. The same may be said of the staphylococcus; it is always present on the skin, but if an animal's resistance be lowered it may gain a footing in the subcutaneous tissue and produce a pustulary eruption. These infections are undoubtedly microbic and also contagious, but



it is very improbable that such infection would take place from animal to animal in the way that the more acute diseases, such as pleuro-pneumonia, &c., are conveyed. Other fatal infections depend largely upon the aid they receive from other organisms, which also are alone practically harmless. For instance, tetanus, if injected in pure culture, would be very unlikely to cause harm, but if it be inoculated with a little garden mould and the many organisms of a saprophytic nature which that contains, the result would be fatal. The seat of inoculation has also a great deal to do with the production of the disease. The fatal *Eastern Cholera*, if injected subcutaneously, does no harm; if, however, it be swallowed, the well-known fatal termination is often the result.

In accordance with the above statement, pathogenic microbes are arranged under these heads—

- I. Obligatory parasites.
- II. Facultative parasites.

Of the first it may be stated that their virulence is sometimes diminished, sometimes increased, but never wholly destroyed, whilst passing through the living organism, as in rabies, where the virulence of the yet undiscovered microbe is diminished whilst passing through the body of the monkey and increased in that of the rabbit. Some of them perish immediately on leaving their host, immediate transmission of the virus being essential, as in rabies, pleuro-pneumonia, and other diseases where inoculation or cohabitation is necessary for the transmission of the disease.

*Obligatory parasites*, more resistant, are preserved for varying periods of time outside of the animal body, without, however, multiplying, and conveyed to healthy animals by various means, such as water, soil, clothing, &c.—contact of a healthy with a diseased animal not being necessary.

*Facultative parasites* are those which, as already stated, live and multiply not only in the bodies of animals, but also in external media,—dead organic matter, water, soil, and even food. Some of these lose their virulence whilst passing through the animal body, and the disease which they induce more or less quickly dies out. We can in this way account for the subsidence, or, as is sometimes the case, the total disappearance, of certain epizootics,—such as epizootic catarrhal fever,

epizootic cellulitis, &c.—and for the sudden reappearance of these diseases. Whilst the majority of these parasites seem to have their virulence gradually attenuated whilst passing through the animal body, they still retain the power of transmitting their pathological effects, when conveyed by a diseased or newly recovered animal from one part of the country to another; hence it may be stated that a disease that has died out in one place owing to its virulence having been reduced in that place, or to the fact that the animals of that place have become immunised, may be virulent in another place, and be conveyed there by some animal which is resistant to the disease, but capable of carrying contagion, and giving it in a virulent form to others not having such resistant power, as was demonstrated by the introduction of smallpox in the West Indies.

It often follows that after a severe and widely distributed outbreak of some epizootic the disease disappears for a long period of time; may it not be concluded from this fact that the germs have a resting stage, and that they are only called into activity by some telluric or atmospheric conditions, the nature of which has as yet escaped detection? Cognisable atmospheric changes have but little effect in calling the dormant virulence into activity, for we witness outbreaks of epizootic diseases in all kinds of weather. Long-continued wet or damp weather has sometimes an appreciable effect, but there are so many exceptions to this that it cannot be said to be a determining condition.

It is already hinted that germs can be held in the air, in water, in the soil—particularly in the superficial layers—foods, stables, byres, sheep pens, vehicles, and utensils, as well as in the various secretions and excretions of animals suffering from their specific effects; hence the importance of the destruction or disinfection of these carriers of contagion.

#### CLASSIFICATION OF MICROBES.

There are various systems of classification,—namely, of Muller, 1773, who established two genera—*monas* and *vibrios*—and grouped them with the Infusoria, but a scientific classification was not made until Ehrenberg in 1838 described four genera, namely:—

- I. *Bacterium filamenti*—straight, rigid.
- II. *Vibrio*— „ —snake-like, flexible.
- III. *Spirillum*— „ —spiral, rigid.
- IV. *Spirochæte*— „ —spiral, flexible.

Dugardin in 1841 united *spirillum* and *spirochæte*, and classed them thus:—

- I. *Bacterium filamenti*—rigid, vacillating.
- II. *Vibrio*— „ —flexible, undulatory.
- III. *Spirillum*— „ —spiral, rotatory.

And up to 1853 bacteria were considered as Infusoria; but Robin then pointed out their affinity to Leptothrix, and Davaine in 1859 demonstrated that they were vegetables and allied to the Algæ, and his conclusions are now universally adopted.

The classification of Cohn is now generally accepted, and he considers that the bacteria are a distinct group belonging to the Algæ, and divisible into four tribes, including six genera.

- I. *Sphærobacteria*—globules (*Micrococci*).
- II. *Microbacteria*—short rods (*Bacteria*).

If the length of a rod be less than twice its breadth, it is considered a bacterium, if longer, a bacillus.

- III. *Desmobacteria*—long rod (*Bacillus* and *Vibrio*).
- IV. *Spirobacteria*—spirals (*Spirochæte* and *Spirillum*).

Cohn noted, notwithstanding the placing of them with the Algæ, that the absence of chlorophyll connected the bacteria to fungi; and Nægeli subsequently adopted this view, and termed them *schizomycetes* (cleft or fission fungi).

We now recognise a group of parasites which has pathogenic members and belong to the group of animal parasites under the zoological family of Protozoa, many of which are pathogenic and cause such diseases as trypanosomiasis, coccidiosis, piroplasmosis, &c.

According to Dr. Stephens's classification, the position of trypanosomes in the animal world is shown in the accompanying table, but this, says Professor Salvin-Moore, must not be accepted as fully proved, for, as yet, all known trypanosomes are *parasitic*, and their origin is unknown, and it is not possible to believe that parasitic protozoa have always been parasitic,

or could be so, and we feel sure that there is, or was, a non-parasitic form, or that they are different phases of some known non-parasitic Protozoa :

### PROTOZOA.

Class.	Sub-Class.	Order.	Genus.
I. Rhizopoda ...	Flagellata	Amœbina	Entamœba. { Cercomonas. Herpetomonas (in insects). Trypanosoma. Trypanoplasma (in fish). Trichomonas. Lambliæ.
II. Mastigophora		Protomastigina	
		Polymastigina	
III. Ciliata } (Infusoria) }		1. Holotricha	Opalina (in frogs).
	Telosporidia	2. Heterotricha	{ Balantidium Paramœcium. Nyctotherus. Gregarina (in meal-worm). Monocystis (in earth-worm). Coccidium Eimeria (in rabbit, cattle, etc.). Hæmamœba (malarial para- sites). Hæmogregarina. Piroplasma. Nosema (in silk- worms). Myxobolus (in fish). Sarcocystis (in cattle).
IV. Sporozoa ...		1. Gregarinida	
		2. Coccidia	
		3. Hæmosporidia	
	Neosporidia	Myxosporidia	
		Sarcosporidia	

A word or two may now be said about the action of microbes : some of them excite fermentation, others putrefaction. The fermentative ones have the power of peptonising and liquefying culture media containing gelatine, and in many instances of producing a poisonous alkaloid—ptomaine—which causes toxic poisoning, a condition distinct from septic infection. The effect of septic poisoning depends upon the dose, a small dose produc-

ing only mild and transient effects, whilst a large one may induce a fatal result; whereas in septic infection a small dose may induce severe disease and death, because the poison introduced is a living organism capable of propagation and numerical increase.

The researches of Brierger have enabled him to isolate various ptomaines from dead bodies, putrid meat, fish, and cheese—*cadaverin*, *putrescin*, *saprin*, *peptotoxin*, and many others, varying in their toxic properties.

Associated with or independent of the production of the poisonous alkaloids, a poisonous albumose is secreted in culture media, and in addition the following substances are evolved:—

- (a.) *Gases*.—Carbonic acid, hydrogen, light carburetted hydrogen, sulphuretted hydrogen, and ammonia.
- (b.) Nitrates, water, and sulphur.
- (c.) Volatile substances (trimethylamin, alcohol, formic acid, acetic acid, propionic acid, butyric acid.
- (d.) Fixed acids,—lactic, malic, succinic, oxalic, and tartaric.
- (e.) Taurin, leucin, alanin.
- (f.) Tyrosin, phenol and kresol (aromatic bodies).
- (g.) Carbo-hydrates, albumoses, peptone, hydrolytic ferments.
- (h.) Colouring matters.

When germs gain entrance into the animal economy, a kind of battle royal takes place between them and a defensive army of phagocytes, consisting mostly of white corpuscles, aided by the connective tissue corpuscles, the endothelial cells of the capillary vessels, soft epithelial and the cells of muscular fibres. These endeavour to arrest the invasion of the microbes by attempting to destroy them. When they fail in this the disease is developed, when successful in their efforts the disease is not developed; when the virulent germs are numerous and strong, they, in a susceptible subject, paralyse and overcome the phagocytes; but when the virus is attenuated, or when the subject is non-susceptible or immune, the leucocytes accumulate around the point of entrance, take up the germs into their substance and digest them; in fact, they kill and eat their enemies.

Again, some germs are antagonistic to others, and it is now well known that the microbes of putrefaction are destructive to those of anthrax; but whilst anthracic blood and tissue are thus

rendered impotent, it does not follow that they are rendered innocuous, for inoculations with such blood have proved that death may result, not from anthrax, but from septicæmia ; and again, the ptomaines of pathogenic germs in many instances, when properly prepared, attenuated, and introduced by inoculation into the bodies of healthy animals, have the power of giving protection or immunity to such animals against the specific effects of the microbes themselves. A knowledge of this fact, first elaborated by Pasteur, has revolutionised modern medicine, and is likely to confer even greater blessings both upon man and the lower animals.

No doubt, when the chemist and the botanist specialise more in discovering the special foods of the saprophytes and the various botanical groupings of these parasites, we shall, in a much better measure, be able to say exactly what processes are gone through by animals which are being immunised from one or other specific disease. I feel sure that this field is not nearly sufficiently exploited by the chemist. I am of opinion that the blood of each individual species of animal differs, and that each of these bloods contains, as identifiable chemical compounds, the various special pabula for the various organisms which give rise to specific disease, and I further think that when any one of these special pabula enters into compound with saprophytes or with other specially prepared chemical compound, that pabulum loses its chemical identity and is so lost, so that if similar organisms enter the blood stream after that event they would be unable to find their special pabulum, and would consequently die without having had the opportunity of causing their own special disease.

One of the most remarkable recent discoveries has been that of "opsonins," and a very clear statement in regard to "opsonins" is contained in Dr. Geo. N. Ross's address delivered recently in Birmingham, and in which Dr. Ross states the following, as taken from the *British Medical Journal*:—

"In the early part of 1903 Wright and Douglas, of St. Mary's Hospital, London, approached the problem of phagocytosis from a totally different standpoint. They first separated the corpuscular from the fluid elements of the blood. That is to say, they obtained leucocytes suspended in a neutral

medium instead of in the blood plasma, and the blood plasma (or blood serum) free from leucocytes or erythrocytes. They prepared an emulsion of staphylococci in normal salt solution, and found that if they brought together only the leucocytes and the staphylococci, practically no phagocytosis occurred, but that the addition of blood plasma (or blood serum) to the leucocytes and the staphylococci effected some change so that phagocytosis did occur. The obvious deduction was that the leucocyte by itself was impotent, and further that the blood plasma contained some substance which was essential to the attainment of phagocytosis.

“Using ingenious methods of their own device, they investigated the blood plasma in order to determine the characters of this phagocytic element, and the following are the most important of their conclusions :

“1. The substance so essential to phagocytosis does not act upon the leucocytes (as a stimulant to the leucocytes, for example), but it combines with the micro-organisms and prepares them for phagocytosis: hence the name *opsonin*, from *opsono*, I cater for, I prepare victuals for. The conception of their mode of action is that the opsonins are carried in the lymph to the nest of microbes which are responsible for the morbid process; that they chemically unite with the micro-organisms, and that then, and not until then, the leucocytes have the power of enveloping and destroying these micro-organisms. Thus it follows that the amount of phagocytosis which is observed is a measure of the quantity of opsonins present in any particular plasma, and does not represent the vital activity of the leucocytes.

“2. The opsonins in a normal serum are almost completely destroyed by heating for ten minutes at 60° C.

“3. The opsonins have been shown to be distinct from the bacteriolysins, the agglutinins, and the antitoxins.

“Moreover, as shown by Bullock and Western, the opsonins have a high degree of specificity. For example, the blood of a person may contain half the normal quantity of opsonins necessary to combat a tuberculous infection, such as tuberculous cystitis, and yet contain a normal amount of opsonins that have to do with an invasion of staphylococci, such as causes furunculosis.

“*Leucocytes*.—Wright and Douglas have shown by a striking experiment how invariable a factor the leucocyte really is. They obtained leucocytes both from an immunised patient and also from a normal individual. To a specimen of each of these they added some normal serum and also some staphylococci, and allowed phagocytosis to take place. They found that in the presence of normal serum the leucocytes of the immunised patient took up just as many staphylococci as the normal leucocytes in the presence of the same normal serum. They next took two portions of a suspension of normal leucocytes to which had been added some staphylococci, and mixed with one of these portions some serum from the immunised patient, and with the other some normal serum, and allowed phagocytosis to take place. They then found that the leucocytes to which had been added the serum from the immunised patient, took up about one-half as many staphylococci as did the leucocytes to which the normal serum had been added. This affords striking testimony that the leucocyte is an indifferent or a constant factor in the phenomenon of phagocytosis. The amount of phagocytosis observed, therefore, represents the quality of opsonins present in the blood. So far as we can tell at present, plasma has nothing to do with the “quality” of the leucocytes.

“*Bacterial Infections*.—Certain generalisations have emerged from the investigation of numerous cases.

“1. If the bacterial infection be strictly localised, the opsonic index of the blood, as concerns the particular microbe causing the infection, is below normal. For example, the blood of a patient who is suffering from furunculosis will probably show an opsonic index of about 0·6 to the infecting micro-organism—that is, to *Staphylococcus pyogenes*; or, again, the blood of a patient who is suffering from tuberculous glands in the neck will probably show an opsonic index of about 0·7 to the tubercle bacillus. In each case, the patient’s blood is compared with the blood of a normal man.

“2. The second generalisation has to do with those infections which are not strictly localised. In such cases the opsonic index will be found high at one time and low at another; that is, the opsonic index in systemic infections tends to fluctuate from high to low. This characteristic is



well shown in cases of acute pulmonary tuberculosis. Dr. Ross has often observed one day an opsonic index 1·6 in such a case, and an index as low as 0·6 a few days later.

“These two generalisations are of primary importance both as concerns the diagnosis and the treatment of bacterial infections.

“*Treatment.*—Briefly stated, the treatment of a bacterial infection, by Professor Wright’s methods, consists in increasing the antibacterial substances of the blood, by inoculating the patient with dead micro-organisms of the same species that has caused and is maintaining the morbid process. This is the general principle.

“The first essential in the treatment of a given case is to learn the particular micro-organism which is responsible for the patient’s infection. In a case of furunculosis, for example, we know that the *Staphylococcus pyogenes* is almost certainly responsible, and so with certain other localised infections the organism is well known. When, however, we have to do with an empyema, or with a cystitis, we only know that one of several organisms is the chief offender. The particular organism is isolated by ordinary bacteriological methods in pure culture and identified. The quantity of those opsonins present in the patient’s blood which have to do with combating this particular micro-organism is then estimated, and is, as a rule, found to be deficient.”

When “opsoninism” is thoroughly understood, we shall have one of the most effectual, or the most effectual method of prophylaxis ever dreamt of.

Many microbes that require a free supply of oxygen are called aërobic, whilst others seem to be destroyed or fail to grow when oxygen is present; these are called anaërobic. Some, however, can live and grow with or without oxygen. Microbes are consequently divided into three groups by Liborius:—

- I. *Obligatory anaërobes*, which flourish in the absence of free oxygen—e.g., *Bacillus butyricus*, and of malignant oedema.
- II. *Facultative anaërobes*.—These grow in free oxygen, but continue to live and multiply, but more feebly, in the absence of it—e.g., Koch’s comma bacillus.
- III. *Obligatory aërobes*, which do not grow in the absence of oxygen—e.g., *Bacillus anthracis* and *Bacillus subtilis*.

*Pleomorphism.*—Lister in 1873 (*Quarterly Journal of Microscopic Science*) indicated that several micro-organisms in their life-cycle exhibit successively the forms of cocci, bacteria, bacilli, and streptothrix. This view is now generally adopted; and Lankester, also in 1873, observed a series of form-phases in the case of a peach-coloured bacterium, which led him to suppose that the natural species of these plants were within the proper limit protean, and that the existence of true species of bacteria must be characterised not by the simple form-features used by Cohn, but by the *ensemble* of their morphological and physiological properties, as exhibited in their life history (CROOKSHANK).

Again, Chauvin has pointed out that the *Bacillus pyogenicus*, by modifying the basis on which it grows, may assume successively the character of a coccus, a long thread, or a spirillum. This theory of pleomorphism is generally but not universally accepted.

It is also stated by some observers that bacteria are not constant in their properties, and the experiments of Nægeli, Davaine, Buchner, and Wernich seem to indicate that both the morphological and physiological characters of microbes are mutable, that changes in the nutrient medium may have effect on their form and size, on their mode of multiplication, and on their physiological and fermentative properties.

Nægeli says that a given bacillus does not invariably produce bacilli of the same structure, and does not always pass through the same developmental stages. A bacterium which under given conditions gives rise to a definite fermentation may lose this property when cultivated under other conditions, and Buchner states that the hay bacillus can be transformed into the *Bacillus anthracis*. He says if hay bacilli are injected into the blood of animals they do not give rise to anthrax. If, however, they are bred for several generations in meat extract, and then in the arterial blood of the rabbit, they acquire noxious properties, and give rise to anthrax in mice after two to nine days' incubation; and conversely, if anthrax bacilli are properly cultivated, they can be transformed into bacilli whose properties are identical with those of hay bacilli. Without entering into the question of the mutability both of form and physiological properties of micro-bacteria, rejected by many observers, it is satisfactory to know that the change in their power of virulence brought about

by cultivation and attenuation, which has already done so much for the prevention and even cure of microbic diseases, will yet do more in assisting in the suppression of many others.

*Period of Incubation.*—The period of latency, or that intervening between the reception of a disease-inducing microbe into the economy and the manifestation of the effect, varies very considerably. Some of the most virulent produce their deleterious effects in a very short time, while others have a prolonged period of incubation. For example, splenic fever and death may be induced in a few hours after inoculation, whilst the deleterious effects of the pleuro-pneumonia contagium, or that of rabies, may not be developed for weeks or even months after introduction into the body. Some contagious poisons again seem to affect one class of animals: thus pleuro-pneumonia is peculiar to horned cattle; epizootic eczema originates in cattle and sheep, but is capable of transmission by direct contact to many other animals, and even to man; glanders affects the horse and ass, but is communicable to man, dogs, sheep, goats, rabbits, and mice by inoculation; and rabies, originating in the dog, is capable of transmission to the majority of animals and to man by inoculation. These peculiarities in the action of morbid poisons are mysterious and unaccountable. We must, however, accept them as facts that investigation may some day throw light upon.

Contagious diseases, whether induced by obligatory or by facultative parasites, may assume an enzootic, epizootic, or even panzootic character, the great feature of which is a tendency to spread rapidly, attacking large numbers of animals in a short space of time, destroying many and incapacitating a large majority.

The term epizootic is derived from the Greek, *Επι*, upon, and *Σῶον*, an animal. Diseases of this order are said to arise from enzootic influences—from *Εν*, in, and *Σῶον*, an animal. Enzootic influences are those peculiar to certain districts, and result from conditions or agencies peculiar to a locality, which favour the development of various miasmatic diseases, such influences becoming epizootic, or affecting the many, from causes as yet unknown. In this order of diseases may be included the catarrhal fever or influenza which prevails more or less at all times amongst the horses of large cities and certain localities. Such diseases may be said to dwell in certain localities, having,

however, the tendency to spread rapidly. They are then said to be epizootic, or even panzootic.

Research during the past decade has been so successful that there have been discovered the causal agent of very many diseases, some of which were believed to be miasmatic, and others, again, to be climatic.

Our knowledge is now so far advanced that we are enabled to divide the causes of all of our known contagious and infectious diseases into three great classes, but this division still leaves us much more matter to be elucidated.

The first class we shall refer to and deal with is that of "The Contagious and Infectious Diseases" due to, as yet, undemonstrable organisms, each of which has the power of producing a toxin which has specific action on one or more tissues and organs of the body. It is probable that when our powers of observation are more perfect, we shall be able to demonstrate the causal agent; but as yet neither can we see it, nor can we filter it out of tissues, fluid or otherwise, in which we are convinced it is present.

Perhaps our hypothesis is wrong, and the causal agent of these diseases may be a diastase such as amygdalin or emulsin. The fact, however, remains that some vital reaction is necessary to produce the substances that cause such serious and fatal results as is seen in diseases which are caused by what are known as ultra-microscopic organisms. But whether that substance is a toxin or a ferment it is impossible to say until the causative organism producing it is demonstrated.

## CHAPTER XIV.

### THEORIES OF INFECTION.

THE method by which contagion or the organisms or virus of an infectious disease gain an entry to the animal's body has given rise to much speculation. It is, however, of no doubt that animals are affected only when their susceptibility to any particular disease is increased—that is, when their natural resistance is lowered. The resistance of the body is due to the fully active condition of that body, and depends entirely upon a certain combination of the cellular and fluid tissues of the blood and organs being in a sufficiently virile state, so as to be able to prevent pathogenic organisms from producing any untoward effects, either by so uniting with them or their poisonous products as to render them inert, or to actually kill and digest these organisms, and so prevent them producing any poisonous properties. Various theories have been introduced to show how this infection or the resistance to it is effected. They are known as the *Cellular Theory*, whose chief exponent is Metchnikoff; the *Humoral Theory* advanced by Ehrlich; and latterly a combination of the two has been demonstrated by Wright, and is known as the *Opsonic Theory*.

Ehrlich explains the *humoral theory* by an ingenious method, showing that in the body there exist molecules of matter which may be acted upon in a deleterious manner by toxins or the bacteria which produce them. These cells are, however, protected by substances which they throw off. The molecule has what are known as receptors, which are ordinarily the recipients of food-stuffs, but which may attract the receptive properties in the toxin or bacterial poison. These receptors may only join with such receptive properties as will

combine with them whether they be the poisonous properties of disease or the natural food-stuffs. If such a union takes place to excess, in the case of disease, then an animal becomes infected. In an animal that has some resistance, this union will stimulate the molecules of matter to the production of fresh receptors, and then in such quantities that they become cast off and free.

In this free state they will still unite with the receptive or haptophore group of the toxin, but, being free, the harmful effect of this toxin is unable to operate, as it is unable to affect the original animal cell which produced the receptor. It is therefore due to the excessive production of receptors that immunity is established, and their excess receptors are the antitoxins.

If by artificial means this immunity is strengthened, the animal's blood may furnish a fluid which, upon injection into another unimmunised animal, will provide that animal also with a temporary immunity, which, upon the excretion of all the fluid injected, will pass away. The theory, when applied to the actual invasion of the blood by bacteria, is somewhat more extended, but the same principle applies. The invading organism possesses a haptophore group which combines with the receptor of the animal cell, and so allows the toxophore, or poisonous property of the organism, to attack and destroy that animal cell. If, however, receptors or side chains are cast off in excess the action is nullified. In this case the receptors are known as the amboceptor or immune body, which has two groups—the cytophile group, by which it joins on to the bacterium, and the complementophile group, by which it joins the alexin, or ferment, and allows of this substance, which is normally present in all animals, to destroy the offending bacterium.

The alexin is a normal ferment in the body, but is unable to act unless the bacterium be joined to it by the action of the amboceptor, or immune body.

The cellular theory demonstrated by many experiments of Metchnikoff explains immunity in a much simpler manner. The leucocytes or phagocytes are the chief factors in the prevention or elimination of infection. If an animal's resistance is high, it is necessary that its leucocytes should be imme-

diately ready to attack and destroy by ingestion and digestion the offending bacteria; if they are unable to do so, then the animal becomes infected with disease.

If the body juices have any bactericidal effect upon the invaders, Metchnikoff thinks that this effect is produced by the action of a product of the leucocytes. The term *negative chemiotaxis* is applied when the phagocytes show a disinclination or inability to attack the bacteria, and a contrary condition is known as a *positive chemiotaxis*.

*The Opsonic Theory.*—Wright has demonstrated another theory by which immunity or infection is accomplished. This is a combination of the above two theories. The phagocytes are the main factors, but they are unable to perform their duties of ingestion and subsequent digestion unless the bacteria have been acted upon by a substance which Wright has called *opsonin*. It is a normal product of the blood, but may be more or less in quantity; if there is a sufficiency, the bacteria are so acted upon and prepared that their ingestion by the phagocyte becomes a simple matter; if they are not sufficient, then the phagocytes cannot act, and a severe invasion of the organism by the bacteria results (see p. 134).

The above is only a very short sketch of the immense subject of immunity, and further reference should be made to the works dealing with this subject alone.

In each of the above theories it is conceded that the immunisation from a disease is the result of the formation of a specific immune body.

Ehrlich thinks that each disease requires the formation in the body of an amboceptor which is specific for only one disease, and if this be not present and the animal still remain immune, the immunity is due to that animal not possessing suitable receptors for that particular disease.

In the cellular theory it is conceded that this is what Metchnikoff calls a *fixateur*, which corresponds to the amboceptor, or immune body, and this also acts specifically to one disease only.

In the opsonic theory each particular disease requires the development of a specific opsonin for that disease. It will thus be seen that the animal organism, according to the above

theories, must, like a chemical factory, be prepared to furnish numerous immune bodies whose function it is to prepare harmful organisms for the fermentative or phagocytic action which is necessary for their destruction.

It must be understood that the above are all theories, although it is possible to demonstrate by the aid of the microscope both the cellular and opsonic theories. The humoral theory can only be demonstrated by deduction. It is very possible that both are correct; and it is a combination of the two that prevents us all suffering from bacterial invasion and extinction.



## CHAPTER XV.

### IMMUNITY.

IMMUNITY may be divided into *active* and *passive*, the former being the result of the immune body in the blood having its origin in the animal itself—*i.e.*, the animal produces it. It is the sequence to recovery from a specific disease, or the introduction of a vaccine. The vaccine is a substance formed of the organisms themselves, and introduced in a weakened or harmless form incapable of producing the disease, but retaining the power of stimulating the animal economy to the production of antibacterial bodies. A *passive immunity* is the sequence of the introduction into the system of immune bodies, and which only remain until in the ordinary course they are eliminated by the excretory organs. The animal is incapable of producing fresh immune bodies itself. This immunity may be accomplished by the injection of an immune serum, and is necessarily only temporary.

It has been found that there are substances connected with the subject of immunity which are demonstrable in the blood serum. The composition of these substances is problematical, but their action upon bacteria is specific.

*Agglutinins.*—It has been noticed that the serum of animals suffering from, or having recovered from, an infectious microbial disease, is capable of affecting the organisms producing that disease in a marked manner. If a mixture of a serum of a typhoid patient be mixed with a young broth culture of typhoid bacilli, these bacilli may be observed under the microscope to gradually collect together into clumps, and lose their mobility. A control is made by preparing the organisms without any serum, but in a solution of normal

salt, and no clumping will be observed. This phenomenon is known as agglutination. The same result may be obtained in many diseases, but it is not so marked as in what is known as "Widal's reaction" in typhoid, and by which a certain diagnosis of the disease may be made. The reaction may also be observed by the action of normal serum when it is undiluted, but its specificity is demonstrated when this serum is capable of producing the result if greatly diluted, and the reaction follows in a comparatively short time. For instance, serum from a typhoid patient diluted in sterile normal salt solution up to some hundreds of times will still cause agglutination in half an hour, but this would not be so in a person whose resistance had not been raised by the stimulation of the presence of the organisms.

Agglutination is therefore an indication that the animal may possess some resistance to the specific infection. The phenomenon may be observed macroscopically in ordinary blood pipettes, and this is probably a better method than the microscopic one.

*Precipitins* are substances like the above, and cause a very similar reaction, which is observed in blood pipettes or tubes, by mixing the organisms with a given dilution of serum and testing the time taken for the organisms to be collected at the bottom of the tube. A sedimentation due to *gravitation* will always show the sediment of bacteria with a concave meniscus, but if by the action of the *precipitins*, the sediment will have a flat upper surface. This method is used largely in the differentiation of varieties of meat and blood. If an animal is injected repeatedly with the blood or muscle of an animal of a different species, its serum will acquire the power of acting upon the blood or muscle juice of that other animal and producing from it a precipitate, by which a definite diagnosis can be made as to the source from which the meat or blood was obtained. It is essential in the application of any of the above tests that a control be made by mixing with the bacteria or fluid to be tested a solution of sterile normal saline, and observing that the agglutination or precipitation only occurs in the tube containing the specific serum, and not in the saline tube.

*Aggressins* are a product of bacteria, and obtained from

inflammatory exudates in the body of an animal. Their action appears to be to paralyse the cellular elements of the blood and to so prevent phagocytosis. They are, as it were, the antipathy of opsonins.

*Bacteriolysins.*—Pfeiffer inoculated cholera vibrios into the peritoneal cavity of a rabbit immunised against cholera; he then at intervals withdrew the fluid from the cavity, and observed the action of the exudate upon the organisms. He found that they lost their motility, became broken up into coccoid forms, and finally disappeared. This is known as Pfeiffer's phenomenon, and is a bacteriolytic action. The substances causing it are bacteriolysins, and may be demonstrated in a variety of microbic diseases in the serum of animals immunised against specific organisms.

These substances are destroyed by a temperature of 55° C., but their activity may be reintroduced by the addition of normal serum from an unimmunised animal, which shows that the heating does not destroy the specific ferment, but only the alexine or complement which is present in all normal blood, and necessary for the union of the amboceptor or immune body to the specific organism so causing the latter's disintegration.

*Hæmolysins.*—It has already been mentioned that if an animal of one species be injected with the blood corpuscles of an animal of another species, the injected animal will acquire the power in its serum of dissolving the red blood corpuscles of the animal with whose blood it has been inoculated. The substance producing this soluble action is known as hæmolysin.

*Cytolysis.*—This—a similar action—is not confined to red cells, but may be demonstrated in the same way with other animal cells, such as spermatozoa. For instance, if the spermatozoa of a horse be injected into the blood of a rabbit, the rabbit's serum will have the property of immobilising the spermatozoa *in vitro*, and finally dissolve them, and the same may be accomplished with leucocytes, &c. The rule as to the application of heat is the same in all these substances—such as agglutinins, precipitins, &c.—as already explained in connection with bacteriolysins. It must be understood that in all such demonstrations it is necessary to hold control experi-

ments—*i.e.*, a tube containing non-immunised serum together with specific substance to be tested, must be treated in the same way as the one containing the immunised serum. In the case of hæmolysins, the corpuscles in the control tube will simply sink to the bottom, but those in the tube containing immunised serum will become dissolved, and a generalised red staining of the fluid will result.

## CHAPTER XVI.

### CONTAGIOUS DISEASES.

#### CATTLE PLAGUE.

*Synonyms.*—*Rinderpest* (G.); *Typhus bovim contagiosus*<sup>1</sup> (L.); *Fièvre pestilentielle du gros bétail* (F.).

*Definition.*—A specific, malignant, and contagious fever. It is indigenous to the Asiatic steppes of Russia, also Hindostan, Persia, China, Burmah, Cochin-China, Thibet, Ceylon, &c., never occurring in this country but as the result of direct or indirect communication between imported cattle (which have been exposed to the contagium) and those of our own shores. It is the scourge of South Africa and of India, but is unknown in America and Australia. The disease has a period of incubation of three days, at the end of which time its local manifestations are developed. It runs a definite course, and usually terminates fatally; but where recovery takes place the animal is rendered unsusceptible to another attack.

It is essentially a disease of the bovine family (the ox, auroch, and zebu), but it may be communicated to the sheep, goat, deer, camel, giraffe, antelope, gazelle, and even the peccary.

It is said that an accidental inoculation of the human skin with the juices of an animal which had died of rinderpest has caused the formation of a pustule similar to that of variola vaccina.—(See Appendix to the Third Report of the Commissioners appointed to inquire into the origin, nature, &c., of the Cattle Plague, page 79.)

<sup>1</sup> The word *contagiosus* was introduced into medicine by Vegetius, and is now recognised as a generic term.

## PATHOLOGY AND SYMPTOMS.

The specific contagium absorbed into the blood gives rise to primary fever. This fever, as indicated by a rise in the temperature, precedes all other symptoms, and occurs in a period ranging from thirty-six to forty-eight hours after an animal has been inoculated. The elevation of temperature varies to some extent. As an average of the normal temperature of the healthy ox during different periods of the day,  $101^{\circ}$  may be accepted. During rumination it may be as high as  $103^{\circ}$ , and may then fall to  $100^{\circ}$ , or even lower. Taking  $101^{\circ}$  as the standard average, it has been found that the temperature rises in seventy-two hours to  $103^{\circ}$  or  $104^{\circ}$ , this elevation of temperature occurring when the animal appears to be otherwise perfectly well. From this we learn that the true period of incubation—latency—is very short indeed, and that, although there are no symptoms visible to the ordinary observer, the disease has actual existence.

Two days after the perceptible rise of temperature has begun, an eruption on, and a peculiar appearance and condition of the mucous membrane of the mouth is seen, having some resemblance at first sight to the appearance of that found in the foot-and-mouth disease. In some rare instances, however, this characteristic symptom has been absent. I have seen it in every case of the plague but one which has fallen under my own observation, and usually veterinary surgeons have been able to diagnose the disease by its presence. Almost simultaneously with this appearance of the mouth, the mucous membrane of the vagina of the cow becomes peculiarly altered. One or other of these signs is rarely absent; so that when they are taken in connection with the elevation of the temperature, the diagnosis of the disease can be made with certainty.

On the day following the eruption in the mouth, or about seventy-two hours after the first elevation of temperature, the animal may be observed to be a little ill, to have less appetite than usual, and to ruminate irregularly. Even at this time, however, the pulse may be unaltered. On the following day, the fourth from the first rise of temperature, the animal, for the first time, shows marked symptoms of illness; and this period, which may be one hundred and ten hours after the real

commencement, is usually considered by superficial observers as the beginning of the disease. The seriousness of the oversight is obvious, on account of the great importance of the earliest possible separation, isolation, or more particularly the slaughter of the diseased animal, and of all cattle with which it may have been in contact.

After the fourth day is over, the constitution is thoroughly invaded. Then ensue the urgent symptoms—the drooping head, hanging ears, distressed look, rigors and twitchings of the superficial muscles, failing pulse, oppressed breathing, foetid breath, and the discharge from the eyes, nose, and mouth.

During the sixth day there occurs a great diminution of the contractile force of the heart and voluntary muscles, the pulse becomes feeble and thready, the respiratory movements are modified, and the animal sometimes shows such weakness in the limbs that it has been thought that some special affection of the spinal nerves must exist. The temperature now rapidly falls, and signs of a great diminution in the normal chemical changes in the body present themselves.

Death usually occurs on the seventh day from the first perceptible elevation of temperature.

Although this is given as the typical course of the disease, there are great deviations from it, as some animals live a longer, many a much shorter time, and the severity and sequence of the symptoms vary considerably.

Rinderpest is caused by an organism of the ultra-visible group. This organism cannot be filtered with undiluted blood; but if the fluidity of the blood be increased by the addition of citrate of soda and normal salt solution, it will pass through a Chamberland filter and the filtrate be as virulent as the blood itself. The virulence is demonstrable before any actual clinical symptoms are noticed, and later all the tissues, fluids, exudates, and excretions of the body are virulent, except the bile, which, as Koch showed, may be used as an immunising agent.

The morbid poison is also contained in the discharges from the mouth, eyes, intestinal canal, &c., of an animal ill from the cattle plague, and that, in a small quantity of the mucus from the nose, eyes, or mouth, when placed in the blood of a healthy animal, increases so fast, that in less than forty-eight hours,

perhaps in a shorter time, the whole mass of blood, weighing many pounds, is infected, and a very small particle of that blood contains enough poison to give the disease to another animal. This at once accounts for the rapid spread of the cattle plague.

It has not been determined to what length of time the blood and textures retain the power of propagating the disease. Professor Jessen of Dorpat, however, says that the mucous discharges, carefully protected, occasionally retain their power of causing the disease by inoculation for no less than eleven months.

The virus, which is both volatile and fixed, can be diffused and the disease communicated by the air for a distance of about fifty yards; but beyond this distance it remains inoperative. It is also conveyed by flies, which, after resting on a sick animal, or its offal, fly about and alight on healthy animals; and by the offal of animals dead of the disease; by hay, bedding, dung, vessels, men, dogs, conveyances; in fact, by any material contaminated by a diseased animal. The contagium is destroyed by dry air, by a temperature of about 140° F., by chlorine, carbolic acid, &c., and, it is stated, by putrefaction.

It is difficult to keep infected blood for any time, and in order to convey blood for experimental purposes, it is necessary that it should be kept in ice, and even then its virulence may not last more than four to five days. It is therefore the more astonishing how it is kept for so long in a natural state. If, however, the infection could be traced, it would be found that it had been carried, owing to carelessness from a more or less recent infection, either by men or insects contaminated with infected material.

The fever, as shown by the elevation of the temperature, begins when the poison has infected the whole mass of blood, or within from about forty to sixty hours after its first entrance into the system. Coincident with the elevation of temperature, the chemical changes in the body are augmented, and, according to Dr. Marcet, one of the products of degradation of tissue—the urea—is largely increased. Soon afterwards the blood is otherwise altered, the amount of fibrin largely increased, the amount of water lessened, and the physical condition of the albumen



altered ; and, according to Dr. Beale, the proportion of soluble substances is also largely increased.<sup>1</sup>

About the third or fourth day local congestions are to be seen on the skin and visible mucous membranes, varying in intensity and in size. Stagnation takes place in the small capillaries of many parts of the body, the blood in which becomes coagulated. A great increase of granular matter is found to occur both within and without the vessels of the congested parts. The

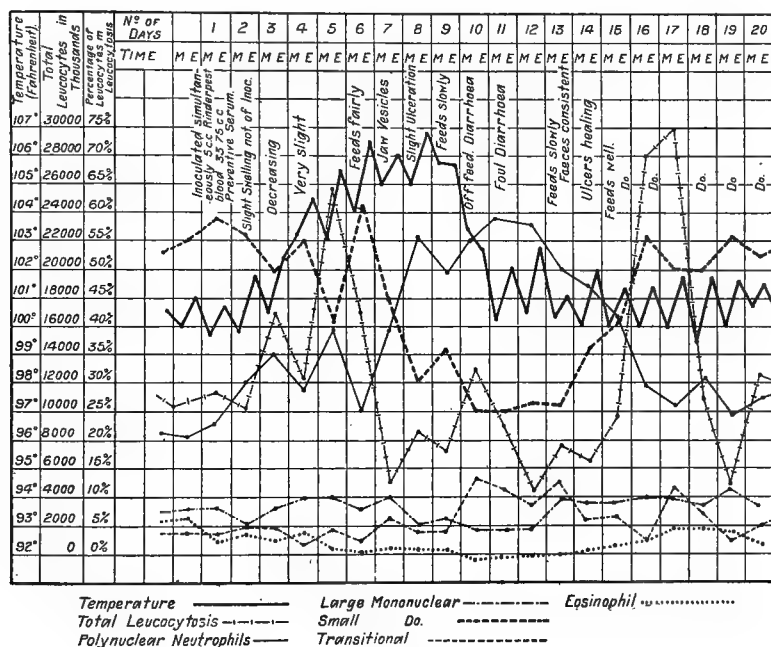


FIG. 9B.

capillaries become greatly enlarged, and the spaces between them lessened or obliterated. At the same time considerable

<sup>1</sup> The following abstract from a Report by Captain Baldrey in 1906 on rinderpest blood is of great interest:

'With regard to the blood in rinderpest observations, a study of the charts (Figs. 9B and 9C) will give a clear idea of the rapid changes which take place in the leucocytic elements. There is a very marked leucocytosis, which may be divided into two periods, the primary and the secondary. The extraordinary feature of this primary leucocytosis is its suddenness. Twenty-four hours after inoculation with rinderpest virus there is an immediate rise to as much as three times the normal, the maximum may not be obtained until the second or third day after inoculation, or concurrent with the first rise of temperature. There is then a fall which is not quite so sudden, and attaining its minimum about the fourth or fifth day—i.e., twenty-four to forty-eight hours after the access of

nutritive alterations take place in the mucous membrane and skin, leading to very rapid and imperfect growth of many of the cellular elements, and followed by rapid disintegration and detachment in the form of discharges. As that portion of the

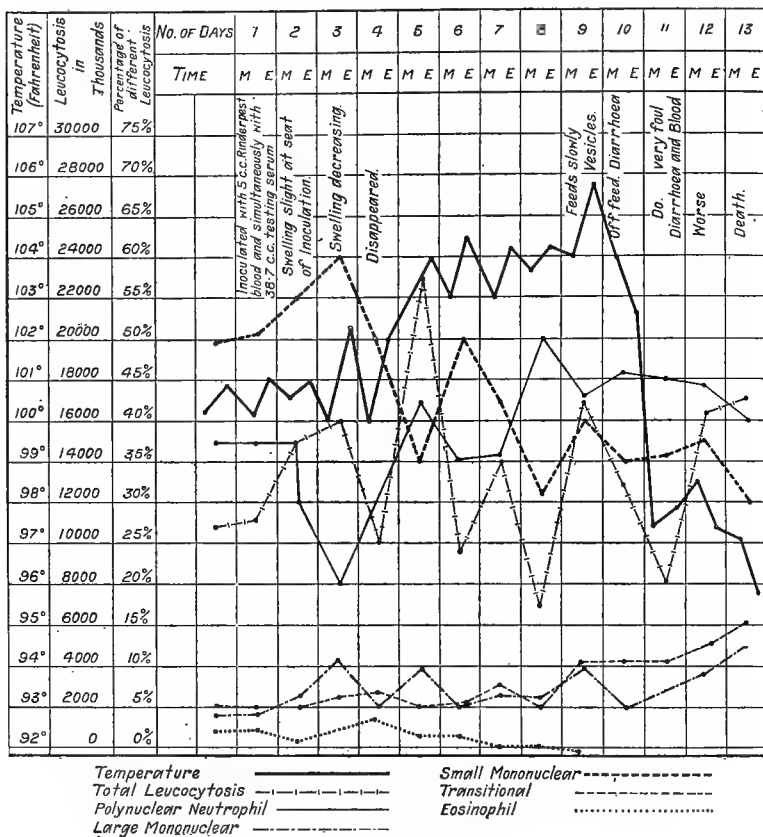


FIG. 9c.

mucous membrane which is most essential for digestion is most affected, the appetite soon fails, rumination ceases, and

the fever. The highest rise recorded was 32,000, and this occurred in a variety of cases on the second, third, and fourth day. The lowest was 18,000. The secondary augmentation is more gradual than the initial, and attains its maximum on the seventh day after inoculation—i.e., the third to fourth day of the fever. This ascent was never so pronounced in an ordinary case of rinderpest as the first rise. The highest recorded was 26,000, and the lowest 17,000. If the animal lives, there will then be a fall, and the secondary minimum will be lower than the initial, reaching, I found, as low as 7,500 per cubic millimetre.'

large accumulations of undigested food are met with in the first stomach. In many cases the villi of the small intestines are so destroyed, that even if food were taken it would scarcely be absorbed in sufficient quantity to maintain life, and hence the rapid exhaustion, failure of the heart's action, depression of the animal heat, and general sinking of the powers. In some cases, when the process is more superficial, the membrane rapidly recovers its healthy condition; and it is curious to find that one affected part may be healing while another is just beginning to suffer.

When, as sometimes happens, the mucous membrane most affected by the congestion is that of the bronchi, the phenomena are not less severe; indeed, the disease is sometimes even more rapidly fatal. A slight cough is soon followed by accelerated breathing, which rapidly increases; and, according to Drs. Sanderson and Bristowe, the difficulty in breathing becomes so great, that some of the pulmonary vesicles are broken, and emphysema, not only of the lungs, but of the subcutaneous tissues of the neck and back, is thus induced. I cannot, however, endorse this, being of opinion that both the pulmonary and subcutaneous emphysemæ are due to the formation of gases in the areolæ of the connective tissue.

With reference to the true nature of the cattle plague, and its identity with or resemblance to various human maladies, many theories have been advanced by writers upon the disease. Some have considered it to be the precise counterpart of typhoid or enteric fever, and this view has been held by the German pathologists for a considerable period, and was undoubtedly the view held by most veterinary surgeons who had the opportunity of witnessing it in this country. Dr. Murchison, however, successfully combats this view, and very lucidly points out its error. He says: "Human enteric fever is characterised by definite and easily recognised anatomical lesions; and for my own part I have been unable to discover any analogy whatever with them in those of rinderpest. The alterations in Peyer's patches in the latter disease are clearly the result of the general inflammation of the mucous membrane, and tend to obliterate the glands, instead of rendering them more prominent. Their resemblance to the lesions of enteric fever is much less than that of the condition of the corresponding glands in cases of cholera, small-

pox, scarlatina, pyæmia, and other blood diseases. It is probable that the diminution in the size of Peyer's patches in rinderpest, as compared with the glands in a healthy ox, may be due in part to the length of time that has elapsed since digestion has been arrested, as it is well known that these glands are larger and more developed during the digestive process than during fasting. But, whatever be the cause, nothing can be greater than the contrast between the appearance of these glands on the tenth day of rinderpest and on the corresponding day of human enteric fever. In the former case the glands have almost, if not entirely, disappeared; in the latter they are enormously prominent, owing to abnormal deposit in and around the glandules." Dr. Murchison quotes the writings of several observers, who agree that the intestinal glands are not enlarged; on the contrary, that they are usually diminished in size, often covered over with a layer of discoloured and softened mucous membrane." "Peyer's glands," says Professor Simonds, in his Report on the Cattle Plague, published in the *Royal Agricultural Society's Journal*, 1857, "are not invariably diseased; but like other follicular openings of the digestive canal, they are often covered with layers of lymph, beneath which ulceration is occasionally observed, but more frequently the surface is healthy, although tinged with blood." After further comparing cattle plague with typhus, influenza, dysentery, erysipelas, scarlatina, and variola, Dr. Murchison concludes that cattle plague has no resemblance to typhoid fever, typhus, scarlatina, erysipelas, influenza, or dysentery, but that it resembles small-pox.

The promulgation of this theory during the prevalence of the plague in 1866 led to the belief that vaccination would prove a preventive, and immediately cattle all over the country were vaccinated, very much to the benefit of the vaccinators but not of the cattle, for it proved a total failure; except, indeed, that it confirmed the view that rinderpest was not variola, but a disease having some resemblance to cholera, scarlatina, and to diphtheria, at the same time having characteristics of its own which separate it from all other diseases, and prove it to be an incurable and highly contagious malady, very properly denominated "the cattle plague."

Mr. John Gamgee, in his work on the Cattle Plague, very

successfully controverts the conclusions of Dr. Murchison, and if the reader is desirous of further information he may consult that work with great advantage.

In addition to the general symptoms of cattle plague which are embraced in the foregoing observations, the more particularly visible signs are worthy of notice; and these are shivering, muscular twitchings, restlessness, often a husky cough, yawning, sometimes great dulness, with drooping of the ears, sometimes excitement approaching to delirium, appetite at first capricious, but soon becoming entirely lost, suspension of rumination, and secretion of milk arrested. The latter symptom was one of great significance amongst cattle owners during the prevalence of the plague, the arrestation being much more sudden and complete than in any other disease. The shiverings and muscular twitchings are not always observable in the earlier stages of the disease, but often seem to usher in the second stage, when the symptoms present a more aggravated character. The animal now incessantly grinds its teeth, arches the back, and draws its legs together, moans, and otherwise shows signs of much uneasiness. The eyes, mouth, and nose are at first dry, hot, and red; the legs and ears are generally cold. At first the bowels are constipated, but this condition is succeeded by violent purging, and the dry condition of the mouth, nose, and eyes is followed by a discharge of a glairy watery character, soon assuming an opaque or turbid nature, which is very typical of the disease. The respiratory movements are generally but not always accelerated; the inspiratory movement is quickened, whilst the expiratory is rather prolonged, and accompanied by a low moan.

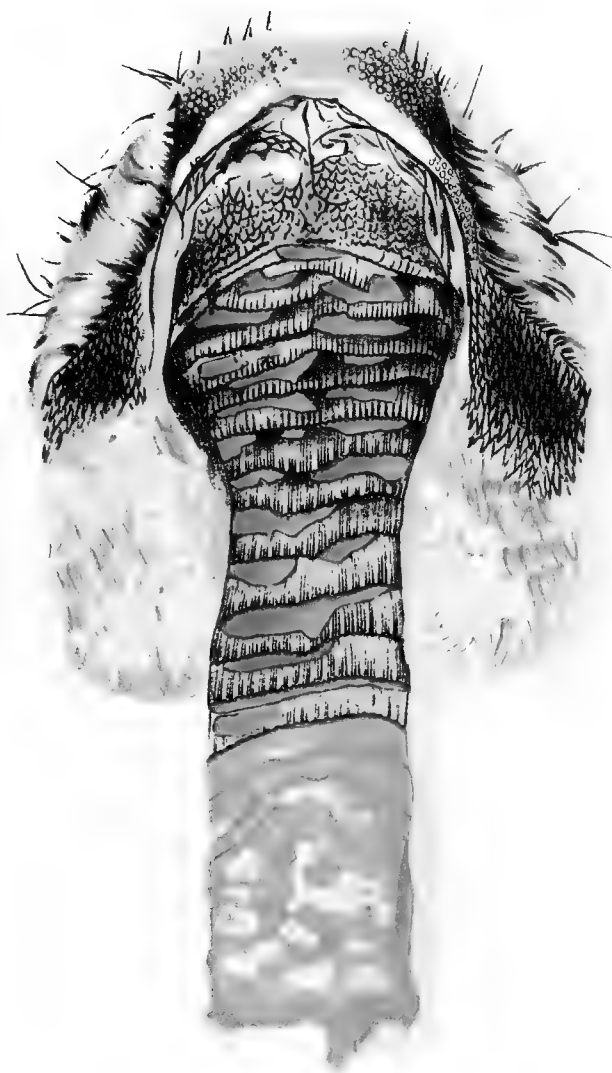
The colour of the visible mucous membranes becomes peculiarly altered. In some cases they present a characteristic salmon-red colour over their whole extent, with deep crimson-red patches interspersed here and there.

Dr. Sanderson, in his Report to the Commissioners, and Professor Gamgee, in his book on the Cattle Plague, quote the observations of Jessen and other Continental veterinarians. Jessen says—"The appearances observed by me on the mucous membrane of the mouth, both in the natural and inoculated disease, are as follows:—In some cases, small round nodules, seldom larger than a millet seed, are observed, which are still covered with epithelium, through which a yellowish or yellowish-

grey material can be distinguished. A few hours later (sometimes not till twenty-four hours) the epithelium gives way, and the contents become visible; hence results a superficial lesion, which, after the removal of the material lying upon it, is scarcely recognisable; it heals in a few days, leaving no cicatrix. In other cases these nodules become confluent, and then give rise to an excavated ulcer of considerable extent, with irregular margins, which, however, usually heals quickly, leaving no cicatrix. In another form of the affection the epithelium is raised in the form of small vesicles, which contain either a clear or slightly turbid fluid, and leave behind shallow, round excavations, with smooth edges.'"

Dr. Brauell of Dorpat, who has made very extensive observations on the anatomy and microscopy of the disease, says that the buccal mucous membrane is usually reddened in patches of greater or less extent; these patches being in some places merely deprived of epithelium, in others covered with layers, varying in size from that of a lentil to that of a fourpenny piece. These layers are of a yellow or greyish colour, and of irregular form; they project from one to two lines above the surface of the reddened mucous membrane, and are so soft that they can be readily stripped off with the tip of the finger.<sup>1</sup> The mucous membrane beneath is more or less reddened by congestion of the capillaries and hæmorrhage, and in some cases broken down in its texture; usually, however, it is entire. Similar alterations are found at the margins of the lips and nose. In addition to these changes, roundish depressions about as large as a hemp seed, their bases covered with a yellowish material, were observed in the mucous membrane of the lips, and sometimes along with them greyish nodules, which might on superficial examination be easily mistaken for vesicles or pustules. On pressing these, a yellowish, semi-fluid mass could be squeezed out, leaving behind one of the depressions or pits above described. These were the appearances seen in animals suffering from the disease naturally contracted. Those seen in inoculated animals did not materially differ. On the third or fourth day after inoculation there was redness of the gum around the incisor teeth, and, more rarely, injection of the mucous membrane of the lower lip. At these spots the membrane was covered with a yellowish-white material, which could easily be stripped off,

<sup>1</sup> See Plate I.



CATTLE PLAGUE.

MOUTH AND PALATE OF A BULLOCK, SHOWING EXCORIATED ULCERS  
AND APHTHOUS DEPOSIT.





leaving small roundish pits. About the same period of the disease, nodules similar to those above described were observed on the lower lip.

“As the disease advances the animal becomes exceedingly restless, lying down and rising again, and otherwise denoting abdominal and colicky pains. Whilst lying down, the head is generally turned upon the upper flank. The voluntary muscles are, in most cases, affected with clonic spasm, and constant twitchings of them occur, more particularly about the neck, shoulders, and hind quarters. Diarrhœa sets in, and the animal becomes in some cases very thirsty; in others, there is loathing both of food and drink. The intestinal discharges, at first black, become of a pale greenish-brown colour, and are very fœtid; and as these are voided they cause much straining (tenesmus) by irritating the rectum. The urine is rather scanty, dark coloured, and sometimes albuminous.” The pulse now becomes much accelerated and very feeble—as high as 120 to 140 per minute; the surface of the body deathly cold; the general weakness increases rapidly, the animal standing with difficulty, and lying most of the time; the cough is weak and soft; the subcutaneous areolar tissue becomes, but not invariably, emphysematus; the buccal membrane and gums are covered with masses of bran-like epithelial scales; the angles of the mouth ulcerated; the vagina of the cow and the preputial orifice of the male present a dark, deep redness. The animal is drowsy and unconscious; the breathing is performed with a spasmodic jerk; the alæ of the nostrils spasmodically open and shut; and as death approaches, the mucous membranes acquire a leaden hue; whilst dark coloured spots of erosion and ecchymosis stud their surfaces. Tympanites sets in, and the discharges from the bowels are involuntarily excreted, whilst muscular twitchings denote the approach of death.

Some cases of cattle plague recover, and in these cases it is observed that the skin over the neck, withers, &c. becomes covered with a yellowish sebaceous secretion, but there are no vesicles or pustules. In some of the fatal cases it is said that there is an eruption: the disease in this respect must, however, differ in different localities.

I have seen recovery take place where subcutaneous emphysema was present over the greater part of the body.

Now and then a relapse takes place after signs of improvement have become established, and in some instances animals apparently quite convalescent commence to purge, and to sink from intestinal and other complications.

Mr. Gamgee says that the disease is periodical in its manifestations: "Improvement in the morning; exacerbations at night; a distinct subdivision of an attack into stages, and from the date of the crisis either sudden aggravation or gradual abatement of alarming symptoms." This periodicity may exist to some extent; it is, however, so slight as to escape ordinary observation. Indeed, in many instances I have observed that the symptoms were quite as bad in the morning as in the evening.

The mortality in Great Britain is always very great. Amongst Russian cattle mild cases of the disease are not of uncommon occurrence, the animals passing through the disease presenting but slight symptoms of it. But even in Russia from 80 to 90 per cent. is reckoned the usual mortality, and under the most favourable circumstances 53 and 56 per cent. have been witnessed.

Although the disease is so highly contagious, it is found that some cattle resist its influence, remaining healthy whilst surrounded with the plague; but it is also no less remarkable that an animal thus exposed to the contagium will, whilst resisting the malady itself, convey the disease to other cattle, the morbid material being lodged about its body.

The *post mortem* appearances of cattle plague vary in different stages.

In the first stage there is congestion of the mucous membranes of the mouth, larynx, pharynx, and particularly of the fourth stomach near its pyloric end, the small intestines are marked with streaks and patches of red, and the follicles are uniformly reddened.

The surface of the mucous membrane is covered with a viscid, tenacious, and bloody secretion; is denuded of its epithelium, whilst the submucous tissue is charged with a turbid semi-fluid exudate. The condition of the first two stomachs calls for no special remarks. Sometimes the rumen presents patches of congestion on its mucous surface, approaching in tint the colour of port wine, and in a very small number of cases sloughing of the membrane has been observed.

The condition of the third stomach has been supposed to give



PLATE II.



CATTLE PLAGUE.

FOURTH STOMACH, PYLORUS, AND COMMENCEMENT OF DUODENUM  
OF A COW, SHOWING CONGESTION AND APHTHOUS DEPOSIT.

*(By permission of J. H. B. HALLEN, M.R.C.V.S., F.R.C.S.E., F.R.S.E.)*

origin to the disease; hence the Germans have called it *Löserdürre*—impaction of the third stomach. The late Professor Dick for a long time held out that the disease was neither more nor less than this impaction of the omasum, that it was consequently a non-contagious disease, and that if by the timely administration of purgatives the stomach could be unloaded, the animal would be restored to a healthy condition. And Dr. Bristowe, in his report to the Commissioners, says, "The contents of the omasum are almost invariably preternaturally dry and caked, and as they are at the same time moulded accurately to the highly papillary surface of the folds of this stomach, it is probably due to the concurrence of these two conditions that the epithelial covering of the folds (when, as often occurs, its normal attachments are loosened) becomes so frequently in this disease removed with the food." Now, I am of opinion that the majority of observers have fallen into some degree of error as to the abnormal state of the contents of this viscus; and have concluded that, because the food is found dry and moulded to the leaves, the condition is unnatural, whereas in reality (as may be confirmed any day by calling at a slaughter-house where healthy cattle are slaughtered) the condition of the contents of the omasum is almost invariably firm, the viscus having the appearance of being impacted.

In some instances the leaves of the omasum are quite healthy; sometimes they are slightly reddened, the vessels which radiate from their attached border being more or less injected, and sloughing may occur in patches.

The fourth or true digestive stomach—the abomasum.—The contents of this stomach are nearly always fluid, and sometimes mixed with blood, and the specific lesions of the disease are intensely marked in this organ. Its mucous membrane<sup>1</sup> is not only intensely red and covered with adhesive mucus, but is studded with numerous superficial erosions, like those which are so common in the ordinary catarrhal inflammation of the human stomach.—(MURCHISON.) The mucous membrane is easily removed from the submucous tissues, and the gastric glands are filled with granular epithelium and with blood. "In addition to the general redness," says Dr. Murchison, "which is most intense in the pyloric region, the mucous membrane at this part often presents circular or irregular patches of a claret colour,

<sup>1</sup> See Plate II.

varying in size from a mere speck to a crown piece. This appearance is due to the extreme vascular injection of the parts in question, and sometimes to actual ecchymosis. The colour may be uniform over the patch; but at other times it is limited to its circumference, forming a coloured rim, with a central greyish-yellow portion. Occasionally these patches may be seen surrounded by a distinct fissure, and in rare cases the membrane corresponding to the patch separates as a slough, which may be found more or less extensively adherent. On separation of the sloughs, deep, excavated ulcers, penetrating the mucous membrane, and even the entire muscular coat, may remain."

With regard to the mucous membrane of the small intestines, it is generally more or less inflamed throughout. Sometimes, however, it has been found almost free from disease. When it exists, however, the inflammation is most intense at the ilio-cæcal opening, and about the middle of the cæcum. The serous surface of the bowels is of a bluish aspect, dotted with spots of ecchymosis, and softened. The discolorations vary much in colour, some spots being scarlet or rose-red, whilst others are of the deepest purple. True ulceration of the bowels is rare, though the mucous membrane is easily removed, and croupous exudates are now and then found in the canal.<sup>1</sup>

The large intestines, according to Professor Simonds (see Report on the Cattle Plague, *Journal of Royal Agricultural Society*, 1857), show marks of the disease even to a greater extent than the small ones. The observations of Professor Simonds were made on the Continent: in this country, the large intestines rarely manifested such signs of inflammation as the small. As already stated, Dr. Murchison has very carefully compared the lesions of the intestinal glands with those occurring in the typhoid fever of man, and has arrived at the conclusion that there is no resemblance. In the healthy ox the glands of Peyer are very often found enlarged and hardened, and when these enlarged glands have been discovered in cattle dead of the plague, microscopic examinations have determined that their contents were chiefly composed of granular masses with cholesterine; products more indicative of a chronic than of an acute alteration.

The lining membrane of the respiratory track invariably presents signs of congestion, and is covered in patches with a soft membranous (croupous) exudation. In the majority of cases

<sup>1</sup> See Plate III.



### CATTLE PLAGUE.

SMALL INTESTINE OF A COW, SHOWING APHTHOUS PATCHES.  
MUCOUS MEMBRANE INTENSELY CONGESTED, VERGING  
TOWARDS GANGRENE.

(By permission of J. H. B. HALLEN, M.R.C.V.S., F.R.C.S.E., F.R.S.E.)

*To face page 160.*





the rim of the glottis is considerably swollen, and the tonsils much enlarged. Emphysema of the lungs is very commonly met with, and is a condition which causes great distress to the animal prior to its death. The remarks of Dr. Sanderson upon this point are very interesting. He says, "During the first stages of the disease respiration is performed in a perfectly natural manner, but about the fifth day irregularities begin to be observable. From time to time the rhythmical movements of the chest are interrupted, and expiration is accompanied by an audible moan. On the sixth day (that is, in fatal cases, the day before death) the breathing usually assumes a character which is so remarkable that if once observed it cannot be forgotten. The chest dilates suddenly, but apparently with considerable effort, in consequence of, as I believe, the unnatural permanent expansion of the lungs due to obstructed expiration. This inspiratory movement is immediately followed by closure of the glottis, the expiratory muscles being at the same time thrown into violent action, much in the same way as they are in the act of rumination. The closure of the glottis is always attended with a sound (such as might be produced by the sudden closure of a soft leather valve) so loud that it can be heard at a considerable distance. This sound coincides with the resisted expulsive effort, which is often so strong as to throw the whole body of the animal into oscillation. The chink of the glottis is continuously closed for one and a half to two and a half seconds, the chest remaining expanded and motionless. At the end of this period the air shut up in the thoracic cavity is expelled with a peculiar grunting noise, which is quite as characteristic as the valvular sound already described. It is immediately followed by a renewed inspiratory effort." Dr. Sanderson says that this peculiarity of movement is the cause of the interlobular emphysema. He says, "The pause, instead of occurring between each expiration and the succeeding inspiration—that is, when the breathing apparatus is in a state of relaxation—occurs when the chest is expanded, or, more accurately speaking, in the middle of the expiratory act; for, inasmuch as the expulsion of air from the chest commences immediately after the chest is filled, a considerable quantity escapes before the glottis has had time to close. At the moment that closure takes place the air confined in the chest is strongly compressed by the action of the

expiratory muscles, and in this way gives rise to the interlobular emphysema, which is so commonly observed in cattle plague." I feel compelled to differ from Dr. Sanderson on this point; my own observations leading me to the conclusion that the emphysematous condition of the lungs is due to the generation of gases in the interlobular lung tissue, and that the peculiarity of the breathing results from, and is not the cause of, the emphysema.

The other organs of the body do not present many *post mortem* signs which may be called peculiar to the disease. The heart is often covered, both externally and internally, with petechial spots.—(See Plate V.) The liver is sometimes pale, sometimes dark; the gall bladder is usually full of bile, and sometimes covered with aphthous patches.—(See Plate IV.) The spleen is usually healthy, a circumstance of great interest, says Professor Gamgee, when taken in connection with the condition of this organ in the typhus and typhoid fevers of man.

The lining membrane of the vagina and uterus, like the mucous membranes of other parts of the body, are deeply tinged with various degrees of redness and purple, and their epithelium is in a softened and semi-detached condition.

The skins of animals suffering from cattle plague present a variety of appearances, such as crusts, eruptions, pustules, and elevations. In inoculated animals dermic alterations are said to be pretty constant, but, as already indicated, they were absent in those cases which fell under my notice whilst the disease was raging in Yorkshire. They were, however, carefully looked for in every *post mortem* examination which I made. Dr. Sanderson says that "they may be described generally as consisting in the first place of *incrustation* of material exuded in a soft or semi-solid state from the glandular follicles of the skin; and, secondly, of pathological changes in the superficial structures of the skin, which, without taking anything for granted as to their nature, may be designated by the term eruption. Subcutaneous emphysema was not infrequent as an *ante* as well as a *post mortem* condition.

PLATE IV.



CATTLE PLAGUE.

GALL BLADDER OF A COW, SHOWING APHTHOUS PATCHES.

*(By permission of J. H. B. HALLEN, M.R.C.V.S., F.R.C.S.E., F.R.S.E.)*



## TREATMENT OF THE CATTLE PLAGUE.

In South Africa, where the late Mr. Duncan Hutcheon, Chief Veterinary Surgeon and Director of Agriculture to the Government of Cape Colony, successfully fought rinderpest and other contagious diseases in animals, there were tried several methods of preventive and curative inoculation. These were as follows:—

“*First.* The inoculation of *healthy* stock with virulent blood in increasing doses, after an injection with virulent bile, with the object of increasing the immunity, but this was found to be unsuccessful.

“*Second.* The inoculation of *infected* herds with serum obtained from diseased animals.

“*Third.* The inoculation of *in-contact* animals with bile from infected animals.

“*Fourth.* The inoculation of *healthy* stock with glycerinated bile.”

The following abstract, from an article written by Mr. Duncan Hutcheon in the *Cape Colony Agricultural Journal*, is most instructive and valuable:—

“Glycerinated bile can with perfect safety be injected into healthy susceptible cattle in doses sufficiently large to give complete immunity against the danger of a second large dose of fresh pure bile communicating the disease. Considering, therefore, that rinderpest has practically died out in the Colony, no farmer with a clean herd would willingly run the risk of introducing the disease amongst his cattle, if he could get them protected without the necessity of doing so. I have no hesitation, therefore, in reply to the question, What method of inoculation should be adopted in the case of clean herds which are in danger? to recommend that they be at once inoculated with a large dose of glycerinated bile (from 20 to 30 c.c.) corresponding to the size of the animals, and follow this inoculation in from eight to twelve days with an injection of a large dose (10 to 20 c.c.) of strong pure bile. If this is properly carried out, such inoculated animals would have a lengthened immunity conferred upon them sufficient for all practical purposes.

“With respect to the doctors Krause’s recommendation to

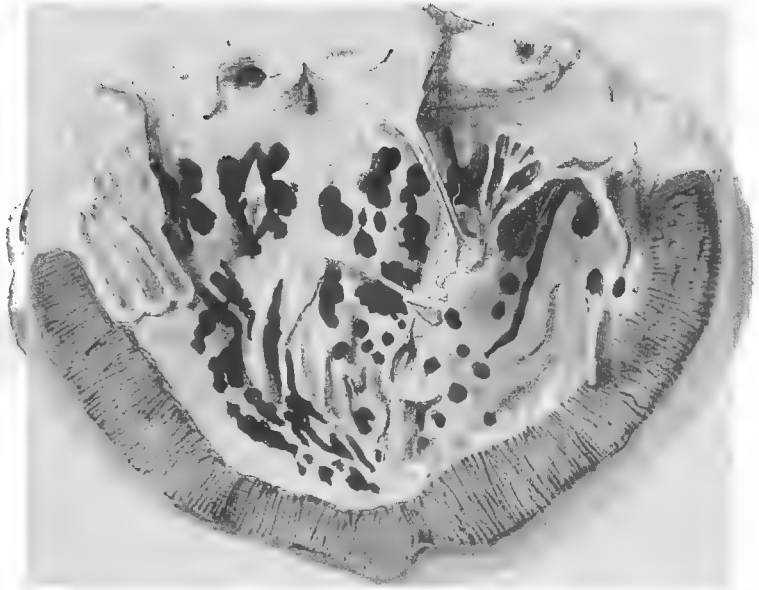
follow these two bile inoculations with an injection of virulent blood—there is no part of South Africa in which an injection of virulent blood after bile inoculation has been so largely practised as in Cape Colony, and our experience is that *one* dose of virulent blood injected on the tenth day after bile inoculation does not strengthen or extend the immunity conferred by the bile, if such bile possessed strong immunising properties, and the blood inoculation that followed produced no fever reaction. If, on the other hand, the bile was weak in immunising properties, the mortality that followed the virulent blood inoculation was very high, in many cases 75 per cent. and even more.

“In controverting our expressed opinion on this point the doctors Krause make a very important qualifying remark. They say: ‘It is an established fact that once an animal is rendered immune, and *gradually infectious* materials are conveyed to its blood, the greater the increase of the immunity will be.’ Quite so: we never disputed that inoculation with virulent blood in *gradually increasing* doses, injected at short intervals after bile, will increase the immunity conferred by the bile. We emphasised this fact (*vide* my annual report for 1897, p. 25). The point that we disputed and still maintain is that *one dose* of virulent blood injected into an animal ten days after that animal was inoculated with Koch’s bile, when the latter confers an immunity which resists the action of the dose of virulent blood so completely that no reaction follows, then the immunity of that animal is not perceptibly strengthened by such an inoculation with virulent blood. I do not think that anyone who reads the account of our experiment at Taaiboschfontein in the Herbert district in 1897 can entertain any doubt on that point (*vide* my annual report for 1897, p. 14).

“But apart from its utility or otherwise, there are very few farmers in the Cape Colony who would now favour the blood inoculation after bile in healthy herds owing (*a*) to the danger of introducing active rinderpest amongst them, and (*b*) the danger of introducing other diseases, such as red-water, by the blood inoculation.

“These are also the chief reasons why the serum and blood method of inoculation should not be applied to healthy herds, in the majority of the cattle districts of the Colony—at least,

PLATE V.



CATTLE PLAGUE.

HEART OF OX, SHOWING PETECHIAL SPOTS.

*(From a Model in Museum of New Veterinary College.)*





not unless the disease should again assume an epizootic form, which we sincerely hope it may not. But independent of the undesirability of introducing the disease into clean herds now that it has become sporadic in its character, it will be impossible to obtain any strong serum after our present limited supply is exhausted, as there will be no suitable animals available for its immediate production. As Dr. Turner remarks, 'such highly fortified animals as would produce strong immunising serum could not be prepared in less than three months. Hence bile must of necessity be used for the inoculation of herds in fresh outbreaks of an isolated and sporadic character.' I would therefore strongly recommend that in every outbreak of the disease that occurs, every drop of suitable bile obtained from the animals which die should be mixed with glycerine in proper proportions, 2 parts of bile to 1 of glycerine, so that it may be preserved and made available for the inoculation of infected herds, and also for the first inoculation of clean herds which may be considered in danger. Pure bile for the second inoculation of clean herds can always be obtained when the disease appears in any locality, which should be the only reason for inoculating clean herds in the immediate vicinity. The method of inoculation which I would recommend in future sporadic outbreaks of the disease is, briefly, as follows :—

*"Infected Herds.*—These should be inoculated at once with either serum or glycerinated bile. Every animal which indicates infection by a rise of temperature should receive a large dose of not less than 100 c.c. of serum, or 30 c.c. of glycerinated bile; the latter should by preference be injected into the jugular vein, so as to secure its immediate action. Then from eight to twelve days after, all the animals in the herd which give no indication of being infected with the disease or fever should receive an injection of pure bile; not less than 10 c.c., and for large animals 20 c.c. This will confer a lasting immunity sufficient for all practical purposes.

*"Clean Herds.*—When it is decided to inoculate a clean herd which is in danger of becoming infected through its proximity to diseased cattle, I would recommend that the animals composing the herd should be inoculated first with 20 c.c. of glycerinated bile, and to follow this inoculation in

from eight to twelve days with an injection of from 10 to 20 c.c. of pure bile. This will confer a strong and lasting immunity on the animals in the herd, and will be free from risk arising from the inoculation or of introducing the disease.

#### DIRECTIONS FOR PREPARING THE BILE.

“The bile should be taken from an affected animal immediately after death, or from one which is killed in the last stage of collapse.

“Biles of all shades of colour—except those which are red from the presence of blood—may be used, so long as they are clear and free from a putrid smell. Thin light yellow biles should also be rejected.

“All the galls extracted at one time should be mixed together, after standing separately for twelve to eighteen hours, so as to render them uniform in strength and immunising properties. Pure bile should be used on the second day after being drawn, unless it is kept in an ice-chest, when it may be kept sweet much longer. But if pure bile is used as a second inoculation only as above directed, it is not desirable to keep it longer than twenty-four hours.

“Glycerinated bile is made by adding 1 part of glycerine to 2 parts of bile, stirring the mixture well; then mix all the biles taken at one time, and allow them to stand for eight days. But if there is urgency, the glycerinated bile may be used forty-eight hours after it is mixed.

“Cleanliness and all antiseptic precautions formerly published, and with which the public are familiar, must be carried out in extracting the bile and inoculating the cattle.”

In countries—such as the plains of India, the steppes of Russia, and Eastern Asia—where the disease is enzootic and has been for thousands of years, cattle have acquired some natural resistance, and the percentage of deaths is not so great as was experienced in England and in South Africa. It is, however, even then as high as 60 per cent., although in many outbreaks only 33 per cent. of animals attacked die; the remainder recover naturally without any treatment. If treatment be attempted, it can only be on general hygienic

principles with nourishing diet and attention to symptoms. It is, however, advisable to endeavour to stamp out the disease by slaughter and isolation, rather than to treat, on account of its extremely infectious nature and very rapid spread.

*Prophylaxy.*—Many attempts have been made to give animals immunity, but it was not until Koch suggested the bile inoculation that any satisfactory results were obtained. This observer found that bile from animals which had died of typical rinderpest was itself incapable of reproducing the disease, but conferred an immunity. It acted, in fact, as a vaccine, the antiseptic properties of the bile destroying the virulence of the disease, but the resultant products having sufficient properties left to stimulate the production of anti-bacterial bodies in the blood of the animal inoculated. The method, however, was somewhat impractical, as it necessitated the using of so many diseased animals, and the bile was not really satisfactory unless obtained on the sixth day of the disease. Various expedients were resorted to in the preparation of this bile in order that it could be kept. The commonest of these was to glycerinate the bile in a similar way to the preparation of vaccine virus. The amount necessary to give immunity was about 10 c.c., and the animal was not immune until about fifteen days afterwards, in which it again resembled the protective vaccines as employed in anthrax, etc. The immunity thus given lasted for some months and could be described as an active immunity. Subsequent experiments showed that a preventive serum could be obtained by immunising animals, bleeding them, and using their blood serum as a prophylactic. The method is to immunise animals with bile and then to increase this immunity by the subcutaneous inoculation of large doses of virulent blood. In a very short time an animal will stand 13,000 c.c. of such blood. Ten days after such a dose the animal is bled from the jugular and will yield about 2,000 c.c. of blood.

The loss will occasion only transient inconvenience, and the operation may be repeated at intervals of a week for three or four times, when it is again necessary to inoculate with virulent blood. This blood is defibrinated and then centrifuged, the supernatant fluid is mixed with 0·5 per cent. of lysol to

insure its keeping aseptic, and it is then ready for use. The doses necessary for the immunisation of cattle vary from 5 to 20 c.c.

Animals vary in the strength of serum they will produce, but it is usual to pool the result of a number of bleedings and to standardise the bulk by testing it on animals. The greatest care is necessary in all these operations to obviate contamination. By the inoculation of this serum alone, a temporary passive immunity only is given, which will last for some ten to twenty days. This is long enough to tide an animal over an outbreak in a herd, but something more is required. It has, therefore, been necessary to inoculate animals with virulent blood and protective serum at the same time. The two are *not* mixed, as this would nullify the action of both; they are therefore inoculated on different sides of the body. A mild attack of the disease is induced, from which the animal usually recovers, and as a result it is given an active or permanent immunity which will last for a year or more. The dose of virulent blood is immaterial, as it has been found that 0.1 c.c. will as surely induce the disease in susceptible animals as will 1 c.c.; the usual dose of virulent blood is 0.5 c.c. The losses from this method are not great—*i.e.*, they should be less than 5 per cent.—and the results obtained quite justify the risk. By the *serum-alone* method, it is obvious that repeated inoculations are necessary, which, in the case of large herds, is both laborious and expensive, although the results may be very satisfactory.

#### RINDERPEST IN SHEEP.

It was at one time affirmed that cattle only were subject to the plague, "*the cattle tribe being alone its victims.*"—(SIMONDS' Report to Agricultural Society, 1857.) Experience has, however, proved that sheep, though less susceptible, are still capable of being affected with the plague.

The symptoms are the same as in the ox, and anyone familiar with the disease in the latter animal will at once be able to recognise it in the sheep. The incubative stage is more variable than in cattle; the disease induced by inocula-

tion appears in from five to eight days ; naturally caused by cohabitation or contact, in from five to twenty days. Sheep kept in fields with cattle suffering from the plague remain a long time unaffected with the disease, many escaping it altogether ; but if kept in closed sheds they are almost certain to become affected in a very short time.—(Professors VARNELL and PRITCHARD, Report to Commissioners, 1866.)

## CHAPTER XVII.

### CONTAGIOUS DISEASES—*continued*.

#### PLEURO-PNEUMONIA CONTAGIOSA.

*Definition*.—An infectious febrile disease, peculiar to cattle and goats, supposed to have originated in Central and been conveyed to all parts of Continental Europe, to Britain, Africa, America, Australia, India, and New Zealand. It is due to a contagium, which gains access to the system by the lungs, and which after an incubative period of from two to three weeks to as many months, induces complications in the form of extensive exudations within the substance of the lungs, and upon the surfaces of the pleura, finally resulting in consolidation of some portions of the lungs, occlusion of the tubes, embolism of the vessels, and generally adhesion of the pleural surfaces. In some cases there is extensive and rapid destruction of lung tissue, with death from suffocation; but most commonly the disease is of a lingering character, symptoms of great prostration manifesting themselves, with blood poisoning from absorption of the degraded pulmonary exudates, and death from marasmus and apnoea.

*Synonyms*.—Lung disease, pleura, new disease, new delight (Yorkshire), pulmonary murrain, epizootic pleuro-pneumonia, &c.; called by the Germans *lungen seuche* and *peri-pneumonia exudativa contagiosa*; by the French *maladie de poitrine du gros bétail* and *pérépneumonie contagieuse*.

#### PATHOLOGY AND SYMPTOMS.

There is much variety in the manifestations of the disease. In some instances, more especially during its first outbreak in a

district, it runs a rapid course, destroying life in the course of a few days, the lungs after death presenting the appearance of a congestive inflammatory change, with embolism and hæmorrhagic infarction. In other cases—and these are the most numerous—the onset, course, and termination of the disease occupy a period of from two to eight weeks, or even longer, the animal becoming much emaciated, and finally succumbing to an exhausting diarrhoea; imperfect aëration of its blood; hydrothorax; the depressing influence of degenerated animal matters absorbed into the blood, and anæmia.

*Premonitory symptoms of the Disease.*—As in cattle plague, the commencement of the disease is often not observable. Thus attention is only called in many instances to an animal for the first time appearing unwell, but in which an examination brings to light the fact that changes of structure have taken place, to such an extent as to convince the observer that disease has been gradually increasing for a lengthened period. The thermometer is not even a true guide by which we are able to discover the end of the incubative and the beginning of the active stage, as later investigations have proved that extensive pulmonary changes may exist without elevation of temperature, and that the fever is probably concomitant with the invasion of the pleural surfaces. For example, the introduction of the disease into Australia from this country proves that the first manifestation of illness is not at all to be depended upon as a guide to its true commencement; for the carriers of the contagion—bulls for breeding purposes—were three months on the voyage, the disease only breaking out after they were landed; but it is of great importance that thermometric observations should be made during the prevalence of pleuropneumonia, in order that owners of stock might be warned in time, and that measures be taken to isolate or otherwise dispose of all animals in which the temperature is found to be rising. In a suspected herd, all animals showing a temperature above 102° should be carefully watched. If the heat rises above this, there can be little doubt that disease is at work. When the disease is established the temperature may rise to 105°, 106°, and rarely to 107° F. In 134 cases, 61 of which were recorded by Mr. Elphick, V.S., Newcastle, and the remainder by myself, only in 8 did the temperature reach 107°.

*The palpable or obvious symptoms* are slight rigors or shiverings, the hair merely standing the wrong way, loss of appetite to some extent, secretion of milk diminished; in some cases the animal "knuckles over" at one hind fetlock, usually the right one; an occasional cough is heard, which is dry and hard in character, not the painful cough of pleurisy, as one would suppose, nor the moist hoarse cough of inflammation of the bronchial mucous membrane; rumination becomes irregular, and although there is some loss of appetite, the animal seems fuller than its fellows, which are healthy and eating vigorously. The bowels are rather constipated, and the urine is scanty and high coloured.

The pulse of cattle, as I have already stated, is not, more especially with regard to its number, a good guide to the practitioner in this or many other diseases; however, as it advances, the pulse becomes accelerated and of a feeble character—sometimes a large soft pulse, other times a small wiry one. These insidious symptoms may continue for several days, the most careful examination of the chest denoting nothing unusual except a tenderness upon pressure applied to the intercostal spaces of one or both sides, and pressure upon the back causing the animal to wince and perhaps give a slight groan. Some cases in an infected herd will at this stage begin to give obvious signs of recovery, and in a few days seem well again, the morbid material having evidently been expelled from the body without causing any extensive pulmonary change. In all cases, however, some amount of irritation and alteration of the lung tissue has been induced, as a cough remains for some time longer. These cases, however, are a source of great mischief, as they become the media through which the virus continues to be conveyed to the cattle with which they are herded, for a considerable period after their apparent recovery. Should recovery not take place, the signs of general disturbance gradually, sometimes rapidly, increase; the cough becomes more persistent, the mucous membranes, except that of the nose, are generally pale, the respiratory movements increased in frequency, more abdominal and shallow; when the animal stands, the elbows are turned out, the nose extended, back arched, and the hind limbs drawn under the body and knuckling over at the fetlocks; when recumbent, the animal throws the weight of its body upon the sternum, and,



owing to the anatomical conformation of this bone and its articulation with the true ribs, the chest is thus expanded.

The breathing becomes painful, and is often accompanied by a moan or grunt, emitted during each expiration, and the nostrils are dilated ; but even these signs are not constant, many animals with extensively consolidated lungs being but slightly distressed in the breathing when left quiet ; but accelerated respirations are easily induced by percussion applied to the sides, or by compelling the animal to move. A discharge sometimes issues from the eyes and nose, which is at first colourless, but often becomes purulent and yellowish, but is seldom profuse. The extremities, as well as the horns and ears, vary much in temperature. Sometimes all are cold ; often, however, one ear and one horn may be cold whilst the others are hot, and so on with the extremities. The surface of the body becomes harsh and dry, the skin appearing tightly bound to the subcutaneous structures, and there is rapid loss of flesh. Constipation of the bowels continues in many instances for a long period ; in others it is succeeded at an early stage by a diarrhoea, which, if not too persistent, seems to have a salutary effect ; but if it assume the colliquative character, the animal dies in from two to three weeks from the first visible manifestation of the symptoms.

The physical signs upon percussion are tenderness and some amount of dulness, the dulness increasing in proportion to the exudation and consolidation. If at the outset of the disease it can be determined that both lungs are inflamed, the prognosis in all cases is unfavourable ; but if, on the contrary, it can be demonstrated that but one lung is affected (the right lung, according to my experience, being more prone to suffer, but showing a greater tendency to recovery), there are some hopes that the case may recover, for very frequently the morbid action is confined to the side primarily attacked.

Auscultation will detect friction and other sounds, which indicate that the disease is not confined to the pleural surfaces ; thus we have crepitations, both large and small, indicative of inflammation of the lung connective tissue, with rhonchus and sibilus, denoting bronchial disease. It is seldom, indeed, but that some sound or other is detectable in all parts, except the lower portions, of the chest ; the consolidation of the lung, extensive though it may be, being insufficient to mask or hide the abnormal sounds emitted by the lung tissue, pleura, or bronchial tubes.

When the disease is confined to one lung, the respiratory murmur in the healthy lung is louder than natural, owing to its having to admit more air than when both are in a state of health. This must not be confounded with a diseased condition; and in order not to make a mistake, percussion must be applied,—the healthy side will be *resonant*, the diseased one *dull*. I have seen some cases where the diseased and consolidated lung has enlarged to such an extent as to push the ribs immediately covering it outwards to some extent, the animal appearing rounder and larger on that side in consequence; and some of these cases have afterwards thriven and become fit for the butcher.

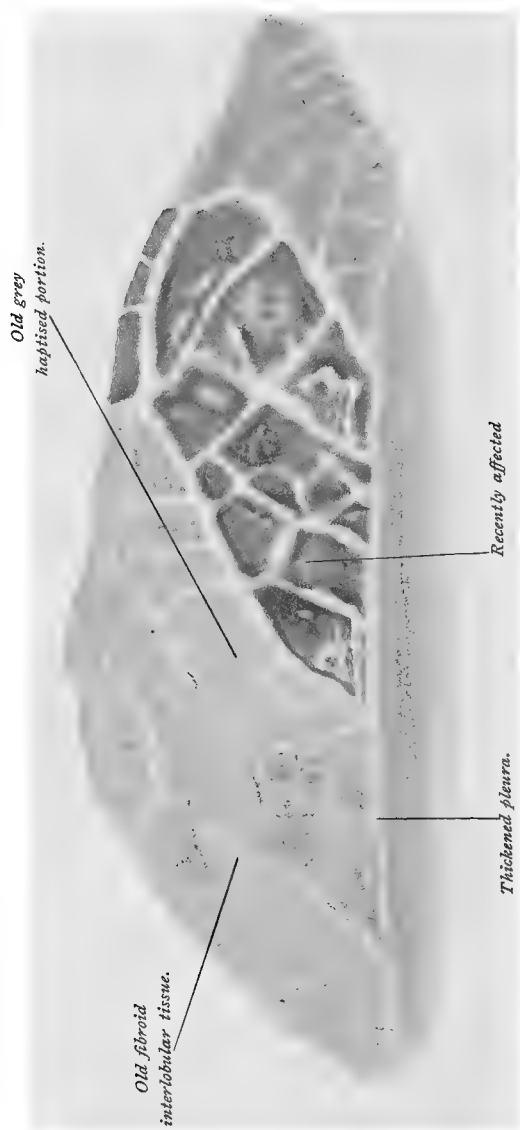
Now and then it is found that some portion of the lung becomes gangrenous, and is coughed up; these cases are, however, very rare. When gangrene occurs, the discharge from the nose is sanious and foetid, and a foetid diarrhoea soon carries off the suffering beast. Abscesses in the lungs are an occasional consequence. An animal apparently recovers from the disease; but after a time begins to lose flesh, and sinks from exhaustion, the *post mortem* revealing caseous tumours or a large abscess or abscesses in the lungs. In some instances of very extensive consolidation, the sounds detectable by auscultation and those emitted by percussion are very trivial. In such it is found that the alteration of structures is most extensive in the central portion of the lungs. One symptom is very diagnostic of this condition—namely, much coughing when the animal attempts to swallow; this is caused by the exudate pressing upon the œsophagus within the chest, and retarding the action of deglutition. When the exudate presses upon the large blood-vessels, there may be turgescence of the jugulars and the venous pulse.

#### POST MORTEM APPEARANCES.

On opening the chest of an animal which has been slaughtered early in the disease, the visible changes are dulness of the affected pleural surfaces; the substance of the lungs, particularly of the large lobes, is red, congested, and more or less consolidated; the redness and congestion being often in patches, each patch being generally surrounded by whitish bands of thickened interlobular tissue, giving them a marbled appearance.



## PLATE VI.



PLEURO-PNEUMONIA CONTAGIOSA.

In other instances the redness is diffused over a more or less extensive surface, and is due to staining of the interlobular bands by extravasated blood, one or several affected lobules forming one patch, whilst the lung tissue for some distance may be more or less healthy, until another patch of more or less consolidated tissue is arrived at. The groups of consolidated lobules vary much in colour, some showing the varying tints of recent inflammation—blood brown, or dark brown redness; whilst others present signs that the exudation is not only consolidated, but is undergoing a change of colour. We have thus the red and a yellowish grey *hepatization*, the red denoting the early, the other the more advanced stages of the inflammation; but the true grey hepatization is seldom or never seen now-a-days, as cattle are slaughtered before the exudate has undergone this change.

In some animals from an affected herd it has been noticed that the first change is a dilatation of the blood-vessels, and the lung assumes a scarlet colour. There is no exudation into the interlobular connective tissue, but the lung is increased in weight, and sinks farther in water than healthy lung.

The *true* grey hepatization seems to indicate a drying up of the inflammatory products, the formation of new connective tissue in the lung, and the disappearance of the alveolar spaces; but the apparent grey or marbling hepatization seen in the early stages of pleuro is due to a distended state of the interlobular lymph vessels surrounding the infarction in the lobule.

The peculiarities in the character of the inflammation of pleuro-pneumonia are its tendency to invade small groups of lobules, more especially those composing the large lobes, or even a single lobule, and to spread not only by diffusion, but also by the invasion of non-contiguous lobules. In this respect it simulates to some extent the pulmonary inflammation induced by the glanders poison, and is characteristic of a local change depending upon a cause existent in the blood; and we may conclude that the true nature of the inflammation differs from that of an ordinary one; that it is secondary to a general infected condition; that the exudates do not tend to become highly organised, but rather to become gangrenous, or

degrade into lower forms of matter—*i.e.*, caseous or calcareous—and rarely to change to cicatricial tissue, and to remain latent in the part for an indefinite period; and that it may commence primarily in the parenchyma of the lungs, and that finally the tubular portion and vessels of the lungs become obliterated, not only by the pressure of exudates external to them, but by histological elements and clots within them. In consequence of these changes, the diseased lung becomes very heavy, sometimes attaining the weight of twenty, thirty, or even fifty pounds, sinks in water, is resistant, solid, non-crepitant, and on section presents the marbled appearance already mentioned.

In those rapid cases of death which occur after the first introduction of the disease into a locality, the changes in the lung tissue are those of an acute character; there is great redness or blackness of the parenchyma—hæmorrhagic infarction—which is at same time loaded with much serosity, and is very friable, soft, and easily broken by the finger; but in the examples which most frequently fall under the pathologist's notice the cadaveric lesions are—*1st.* Extensive deposits of yellowish, friable layers of false membrane upon the surface of the pleura and upon the pericardium. These false membranes exist upon one or both sides, as the case may be. Portions of the lungs are found adherent to the sides and to the diaphragm, but in many cases the bands of lymph are of a friable nature, the adhesions very imperfect, and the opposing surfaces easily separated, while there is, in old-standing cases, generally much fluid in the cavity of the thorax, in which flakes of fibrin are seen floating. The effused fluid is of a yellowish colour, contains much albumen, and if exposed to the atmosphere will often coagulate into a gelatinous clot. There is much variety in the quantity of the fluid. In some cases the pleural cavity and pericardium will contain several gallons, whilst in others the quantity may only measure a few ounces.

The pulmonary pleura, in addition to the bands of lymph, which form the adhesions already alluded to, is invested by a firm layer of lymph, which may be stripped off, leaving the lung rough, mottled, and having papillæ-like eminences upon its surface. These bands of lymph, as well as the flakes found floating in the serum, are composed of fibrin. When examined

microscopically, cellular elements, partaking of the nature of pus cells and white blood globules, are seen imbedded in the fibrin, which is sometimes granular, but often filamentous. The turbidity of the serum is caused by these globules and some amount of fatty matter.

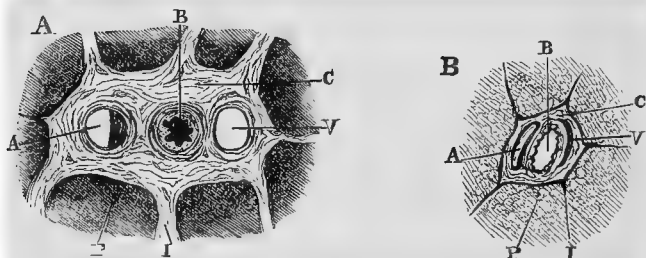
The tissue connecting the pleura to the thoracic walls is but slightly involved, and when removed after slaughter, the underlying structures seem healthy, but a careful examination of the part will show the ribs to be bare, stripped of their pleural covering, and to have an unnaturally clean whitish appearance, and the intercostal flesh, in advanced cases, unhealthy, moist, or even dropsical. The pericardium is also covered with flakes of lymph, and greatly thickened by exudative materials.

It has been considered that the primary seat of the disease is situated in the pleura; that the inflammation is of a rapidly spreading character, causing the formation of what have been termed by Rokitansky "the croupy exudates," both upon the surface of the serous membrane and in the substance of the lungs. As a rule the inflammation in pleuro-pneumonia contagiosa is more pronounced in the parenchyma than upon the pleural surface; but this is liable to exceptions, for in some cases the pleural disease is more marked than the parenchymatous. Some writers think that the disease may begin in the bronchi and air cells, and that the irritation extends from the tubes and cells into the parenchyma and pleura. Their conclusions are based upon the fact that inflammatory products are generally found, in the advanced stages of the disease, in the air vesicles, blood-vessels, and bronchi, as well as in the other portions of the inflamed part.

Dr. Yeo, in an elaborate report published in the *Veterinary Journal* and other periodicals, concludes that the disease originates in the bronchial tubes, and that the surrounding tissue is in a state of chronic inflammation for a lengthened period before any symptoms are manifested. He says—"I am convinced that the lung disease usually exists for months without being suspected, and invariably the beast is first thought to be sick only when the affection has spread to the pleura, and caused intense inflammation of that membrane with its accompanying well-marked symptoms."

These very confident conclusions of Dr. Yeo, if allowed to go unchallenged, are calculated to lead to much misapprehension, and even to actions at law. They prove to me that he has had but little or no opportunity of studying the disease in the living animal, and that he bases his conclusions upon what he has observed in the laboratory.

Now, if pleuro-pneumonia were a species of bronchitis, or, as he puts it, if the virus acted primarily on the bronchial mucous membrane, one would naturally conclude that the bronchial walls would in the earliest stages show evidence of disease; but such is not the case, and only occasionally do we meet with congestive patches along their course in those parts of the lungs already involved in the disease. As the disease advances, however, the bronchi, as shown by Dr. Yeo in the woodcut,



FIGS. 10 and 11.—Transverse section of Broncho-vascular System, contrasting the healthy with the diseased state.

A. State of advanced disease. A. Artery, partially occluded by a thrombus. B. Bronchus, contracted and plugged. V. Vein. C. Common broncho-vascular sheath, thickened by exudation. I. Interlobular tissue. P. Lobular parenchyma.

B. A corresponding broncho-vascular system in health.<sup>1</sup>—(Yeo.)

<sup>1</sup> The cut was kindly sent me by Dr. Fleming.

become occluded, their walls invaded by the inflammation, and they, as well as the blood-vessels, alveoli, and air cells, are filled with a solid exudate, differing, however, from that of bronchitis, where the occluding material is composed of more or less fluid catarrhal products; and one most important condition is absent, namely, the blood-vessels of the inflamed lobules are not occluded by coagula as in pleuro-pneumonia contagiosa. In some instances, rarely met with since early slaughter has been made compulsory, the bronchial tubes of a non-invaded



lung territory are found occluded, and the lung tissue surrounding them collapsed by the gravitation or entrance into them of the degraded, softened products of the pneumonia, which, having escaped from the seat of formation, have been forced by the inspiratory act into the healthy tubes. Here we certainly find the bronchi filled with diseased products, and the pleural surface presenting little or no signs of disease; but an examination of the pneumonic products will at once prove that they consist of degraded fibrinous exudates, and that the parenchymatous disease is antecedent to the occlusion of the bronchi. In the second place, Dr. Yeo concludes that, in the common run of cases where pleurisy is associated with extensive disease of the lung, the latter gives the impression that it is of much older standing than the pleural affection. He says, "The pleurisy is commonly acute; while in the lung we usually have evidence of such chronic change as would require a very long time for their development."

If Dr. Yeo had had clinical experience amongst cattle he would have seen that these conclusions are quite opposed to facts. Many instances have occurred in my experience where animals, apparently healthy, which had been slaughtered in consequence of having been in contact with others affected with pleuro, have presented, *post mortem*, round patches of consolidation in one or more parts of their lungs, always associated with pleural exudation, and sometimes even adhesion of the opposing pleural surfaces; indeed, in some instances the most pronounced evidences of the presence of the disease have been patches of pleural exudation with but little or no corresponding invasion of the lung parenchyma.

Then, as to the chronicity of pleuro-pneumonia, the statements of Dr. Yeo are against all clinical facts, and consequently must not go unchallenged. A reference to the accompanying woodcut (fig. 12), and a short history of the case from which it was obtained, will illustrate this point. The large dark patches reveal the condition of dark red consolidation, and the smaller ones the commencement of that process, which is evidently that of hæmorrhage into the alveoli; the dark patches are surrounded by œdema of the tissue contiguous to the points of extreme congestion; the interstitial tracts are much distended, and if

carefully cut, a quantity of straw-coloured lymph, only found

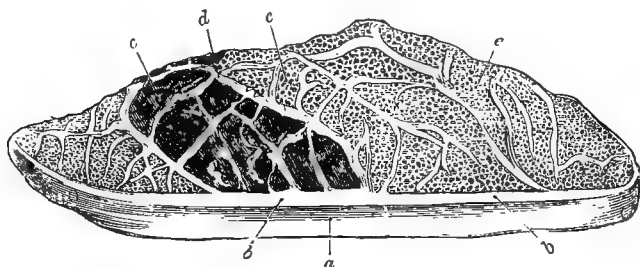


FIG. 12.—(a) Pleural surface presenting a smooth opaque appearance. (b) Subpleural exudation. (c) Red consolidation. (d) Thickened interlobular tissue. (e, e) Hæmorrhagic spots, due to extravasation into alveoli.

in recently invaded lung territory, flows from the cut surface. This may be collected and used for inoculation purposes. At a

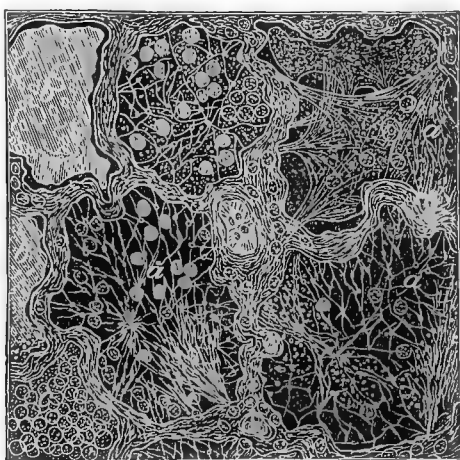


FIG. 13.—(a, a) Exudation in air vesicles, composed of a fibrinous network, with leucocytes lying in it. (b, b) The same caseating. (c) Air vesicle filled with leucocytes only. (480 diam.)

In the centre is seen a blood-vessel filled with a fibrinous plug, and around it numerous leucocytes in the substance of the wall of air vesicle.

later stage this is more gelatinous in consistence. The pleural

surface is much thickened, the exudate being both within and upon the serous membrane. Microscopically examined, as shown in fig. 13 the diseased products have every appearance of being of recent origin, the secondary or degenerative changes (*b, b*) which occur very early in pleuro where the blood supply is cut off by plugging of the blood-vessels, having only commenced. The history of the animal from which the specimen was obtained was clearly traced. It had been brought from a healthy herd six weeks prior to slaughter, thrived and milked well up to the two days prior to slaughter, when it presented slight signs of illness, became rapidly worse, and when examined clear evidence was obtained that both lungs were gravely invaded. I may state that, along with four others, all of which succumbed, the animal had been brought into a byre where the disease had previously prevailed, but which had been declared free under the Act of 1868. Taking into consideration the clinical fact, which has forced itself upon me upon many occasions, that it generally takes from a month to six weeks for the disease to develop in a healthy animal introduced into an infected byre, and the condition of the diseased products in the above case, one cannot help concluding that pleuro-pneumonia is often a very acute disease, and that it may rapidly involve large tracts of lung tissue.

In other instances, however, the invasion is confined to small areas; the disease may then assume a mild type, and the animal recover; whilst in others the disease becomes chronic, the animal dying, not so much from destruction of lung structure, but from a mal-condition of the whole system, arising from absorption of the degraded products of the inflammation.

The *cause* of pleuro-pneumonia, in this country at least, is undoubtedly contagion and infection, and these only. My experience of the disease enables me to state that no mismanagement with regard to feeding, housing, or the general treatment of stock, will induce an outbreak of pleuro-pneumonia contagiosa.

#### PREVENTION OF PLEURO-PNEUMONIA.

When the disease breaks out in a herd, the affected animals are to be isolated, or, if "in condition," slaughtered for beef, experience having proved that such beef is good and wholesome if the animal is killed early in the disease. If the animal be

out of condition, slaughter and burial are the safest, and, in the end, the most economical; indeed, it may be distinctly laid down that the slaughter of the whole herd for human food is the cheapest in the end, the hides, hoofs, &c. being carefully kept from coming in contact with any healthy cattle. Some recommend the destruction of hides, hoofs, &c. I think this a wasteful destruction of property, and fail to see how the disease can be propagated by them; unless, indeed, they are actually brought into contact with healthy cattle—not a very likely occurrence.

*Disinfection.*—All sheds, cow-houses, or other premises which have contained cattle affected by the lung disease should be thoroughly cleansed and disinfected. The best and cheapest way of doing this is—1st. To burn sulphur in the buildings; 2nd. To whitewash the walls, stalls, roofs, and every *nook* and *crevice* within the building with lime-wash containing carbolic acid (crude), in the proportion of one pint to each bucketful of whitewash.

*Inoculation of pleuro-pneumonia products* was attempted by Dieterichs with no satisfactory results. The experiments were repeated by Vix, who obtained results in the form of pneumonia, which was due in all probability to pyæmia. Nothing conclusive, however, was obtained until Dr. Willens of Hasselt, Belgium, instituted a series of experiments, the result of which led him to arrive at the following conclusions:—

“1. Pleuro-pneumonia is not contagious by inoculation of the blood or other matters taken from diseased animals and placed on healthy ones.

“2. That the blood and the serous and frothy liquid squeezed from the lungs of a diseased animal in the first stage of pleuro-pneumonia are the most suitable matters for inoculation.

“3. The inoculation of the virus takes from ten days to a month before it manifests itself by symptoms.

“4. The matter employed for the inoculation has, in general, no effect upon an animal previously inoculated or having had the disease.

“5. The inoculated animal braves the epizootic influences with impunity, and fattens better and more rapidly than those in the same atmosphere with it that have not been inoculated.

“6. The inoculation should be performed with prudence and circumspection, upon lean animals in preference; and towards

the tenth day after the operation a saline purge may be given, and repeated if necessary.

"7. By inoculating pleuro-pneumonia a new disease is produced; the affection of the lungs, with all its peculiar characters, is localised in some part on the exterior, but whether it occasions apparent morbid manifestations or not, the inoculated animal is preserved from pleuro-pneumonia.

"8. The virus is of a specific nature; it does not always act as a virus. The bovine race alone is affected by its inoculation, since other animals of different races, inoculated in the same manner, and with the same liquid, experience no ill effects."

*Method of inoculation.*—The virus is to be selected from the interlobular tissue of a lung in the first stage of a mild attack. The lung will then be found distended with a yellow semi-fluid exudate. All parts of the lung which present any appearance of dark red colour, and are consolidated, or which have the appearance of being gangrenous, should at all times be avoided. The selected portion of lung, after having been cut along the lymph spaces to allow the fluid to ooze out, is to be placed whilst warm in a strainer, over a clean stoneware or glass bowl, covered over by flannel or cloths, to keep in the warmth and to prevent dust, &c. gaining access. In a short time a quantity of clear yellow fluid will be obtained, which, if not required for immediate use, is to be enclosed in glass tubes, about four inches long, three-eighths of an inch in diameter, and, when filled, hermetically closed by a blow-pipe flame.

The tip of the tail is the spot which should be selected for inoculation, and a single drop of the virus is sufficient. It is better to inoculate on the upper than on the lower surface of the tip of the tail, removing the hair, and then slightly scarifying the skin, the scarification being quite superficial. The point of the tail is selected in preference to any other part, because it can be easily amputated if gangrene occur.

In the most favourable instances, a slight heat and swelling occurs round the inoculated spot in a period varying from a week to two months; generally, however, the eruption manifests itself from the ninth to the sixteenth day, accompanied by slight rigors, loss of appetite, and slightly diminished secretion of milk. When

the operation has been properly performed, and the virus carefully selected, the effects are generally as above described; but when the virus is putrid or badly selected, or, as sometimes happens, some peculiarity exists in the inoculated animal, the primary swelling is excessive, the tip of the tail becomes gangrenous, the animal suffers from a high state of fever; secondary deposits occur at the root of the tail, around the anus, and in the abdominal glands, and death occurs in a few days after the inoculation.

The conclusions arrived at by the Belgian Commission with regard to inoculation are as follows:—

“ 1. The inoculation of the liquid extracted from the lungs of an animal affected with pleuro-pneumonia does not transmit to healthy animals of the same species the same disease—at all events so far as its seat is concerned.

“ 2. The appreciable phenomena which follow the inoculation are those of local inflammation, which is circumscribed and slight on a certain number of the animals inoculated; extensive and diffuse, with general reaction proportioned to the local disease, and complicated by gangrenous accidents, on another number of the inoculated animals, so that even death may result.

“ 3. The inoculation of the liquid from the lungs of an animal affected with pleuro-pneumonia exerts a preservative influence, and invests the economy of the larger number of animals subjected to its influence with an immunity which protects them from the contagion of this malady during a period which has yet to be determined.”—(GAMGEE.)

The losses sustained during the experiments of the Commission amounted to 11·11 per cent.; the number of animals on which the operation was benignant was 61·11 per cent.; the proportion in which there was gangrene and loss of a portion of the tail was 27·77 per cent.; in twenty-one subjects the inflammation was very severe, and complicated by gangrenous phenomena, causing the death of six; and lastly, the recoveries amounted to 88·88 per cent.

Following the method of Willens, Professor John Gamgee introduced preventive inoculation into Edinburgh about 1857, but the casualties were very numerous, many animals dying from blood-poisoning induced by the operation; in fact the

operation was condemned as being worse than the disease. In London it was condemned by Professor Simonds and others, and to the present time it finds no favour there.

Dr. Burdon Sanderson and other experimentalists recommend the injection of the lymph into the venous system by means of a small syringe, selecting the superficial aural vein for that purpose. This method of inoculating, while inducing no local results, is said to be quite as effective as that of inoculation of the tip of the tail.

Further investigations on the cultivation of virus, as recommended by Pasteur with those of anthrax and fowl cholera, may reveal the fact that an attenuated virus is quite as effective as that removed from the pneumonic lung, and that successful inoculation by direct injection into a vein may be performed without inducing any local ill effects.

Intravenous injection has also been investigated by Professors Thiernesse and Degive, who, having found that inoculation with the lymph of pleuro-pneumonia in the cellular tissue of the dewlap generally caused death, performed a series of experiments, and concluded—

1st. That the injection of pleuro-pneumonic virus into the veins is not at all dangerous, if care is taken that not a single drop of this liquid falls into the cellular tissue.

2d. That this infusion possesses the same properties as caudal inoculations,—that is to say, that it invests the animal body with a real immunity.

Professors Thiernesse and Degive inject the virus into the jugular, and recommend certain precautions to prevent all contact between the virulent fluid and the cellular tissue. If, however, the steel cannula be plunged into a vein, and then the syringe adapted carefully, and care taken that all the fluid is injected into the vein before withdrawing, first the syringe, then the cannula, there will be little or no danger of this occurring.

These various methods of preventive inoculation have been within the past twenty years much improved upon.

Firstly, the collection of the lymph is made with much care. A lung, a portion of which has only an exudate of lymph into its interlobular tissue, but which has little or no

hepatization, is selected; an incision is carefully made through the pleura into one of these spaces; the trabeculæ are then broken down with a blunt spatula, to enlarge the cavity; the lymph, as it accumulates therein, is carefully removed by means of a porcelain spoon and placed in an aseptic wide-mouthed bottle.

The lymph thus collected is allowed to stand for at least twenty-four hours, during which time it diminishes in virulence, and does not cause such a strong reaction when inoculated into the tail. Such lymph causes marked lesions in the tail, and a certain amount of systemic disturbance within six days under ordinary conditions amongst cows in byres, but takes two or three days longer in cold weather and in stock out at pasture.

If the lymph be introduced into the under or upper surface of the tip of the tail, at the expiry of about six days, the hairs will stand on end, and the part become swollen. This swelling is very typical, has the appearance of brawn, and glistens like jelly.

The wound caused by the inoculation should heal by first intention in a successful inoculation. Should it not do so, but commence to suppurate, the operation is usually unsuccessful. So long as the swelling remains local, and only the last 6 or 8 inches of the tail are affected, things may be considered to be favourable, and no interference is necessary; but if the swelling should commence to extend up the tail and become diffuse, then it will be necessary to amputate the tail *above* the swelling, taking great care that all invaded tissue is removed.

The virus causes thrombosis of the vessels, and if the incision of amputation be through infected tissue, there will be no *spurting* of arterial blood.

By the Contagious Diseases (Animals) Act, 1878, it is enacted that all cattle suffering from pleuro-pneumonia are to be immediately slaughtered; and by the Pleuro-Pneumonia Slaughter Order of 1888, all cattle being or having been in the same field, shed, or other place, or in the same herd, or otherwise in contact with cattle affected with pleuro-pneumonia, are to be slaughtered within ten days after the fact of their having been so in contact has been ascertained, or within



such further period as the Privy Council may in any case direct. All cattle which have been certified by an inspector of the Privy Council to have been in any way exposed to the infection of pleuro-pneumonia are also to be slaughtered within such period as the Privy Council may direct.

#### THE PATHOGENIC MICROBE OF PLEURO-PNEUMONIA.

At the meeting of the National Veterinary Medical Association held in Edinburgh in 1886, I stated that pleuro-pneumonia was due to an organism. This announcement was received with derision by certain members present. At that time, after several years' work, I had found two organisms, a bacillus and a micrococcus, and was of opinion that the micrococcus was the pathogenic microbe. The question has been since studied by various Continental observers. Lustig found that there are generally four organisms in the exudates of pleuro. These four, however, may be classified into two groups, as seen under the microscope:—1st. A bacillus which rapidly liquefies gelatine in cultivation tubes: 2nd. Micrococci, which are proved by cultivation to be of three kinds—(a.) a micrococcus whose colonies are white, like boiled white of egg; (b.) a micrococcus the cultivations from which assume a golden yellow colour; and (c.) a micrococcus whose cultures on gelatine resemble drops of wax. These four organisms were studied by Lustig in 1885, and by Cornil and Babes in 1886, but the results which the latter obtained were not very precise; and they concluded that their work, and that of all investigators who had preceded them, would have to be repeated.

Arloing and others have also found four different organisms:—1st. A bacillus called by Arloing *Pneumobacillus liquefaciens bovis*; 2nd. Three micrococci—(a.) a non-liquefying coccus whose colonies resemble drops of wax—*Pneumococcus guttaceri*; (b.) a micrococcus the white colonies of which spread in a thin layer, and as this grows older it becomes wrinkled and folded—*Pneumococcus lichenoides*; and (c.) a micrococcus whose colonies, elongated or round, assume a beautiful orange tint—*Pneumococcus flavescens*. Arloing is now of opinion that the *Pneumonia bacillus liquefaciens* is the pathogenic microbe of pleuro-pneumonia; for, by means of subcutaneous inoculations of cattle with pure cultivations of the microbes, it was

found that the bacillus produced the greatest effect, and when injected in larger doses pulmonary effects were induced closely resembling those of pleuro-pneumonia. Arloing has repeated his experiments, and says—"I have reproduced in the bovine with pure cultures of the pneumobacillus taken between the second and tenth generations the typical alterations produced under the skin or in the chest by the virus of the peripneumonia contagiosa. I then emphatically say—(*first*) that the virulent agent of contagious peripneumonia is an ordinary microbe; (*second*) that this microbe is the *pneumobacillus liquefaciens bovis*."—(*Compte Rendus d'Académie des Sciences*.)

Nocard has shown that all the above observations are wrong. The casual organism is what has been described as an ultra-microscopic one, as it is too small to be seen definitely with the strongest lenses. Nocard demonstrated its presence by obtaining pure cultures in bouillon contained in celluloidin sacs and placed in the peritoneal cavity of rabbit. By this means a growth was obtained in the broth which rendered it cloudy, and it was found capable of reproducing the disease and being again recovered from the lungs of infected cattle. The organism would not grow on ordinary media in the incubator, and, being so small, its morphology could not be made out. This organism may be taken conclusively as the *causa causans* of the disease, and pleuro-pneumonia contagiosa can now hardly be placed among that class of contagious diseases which is described as being due to organisms of the ultra-microscopic variety.

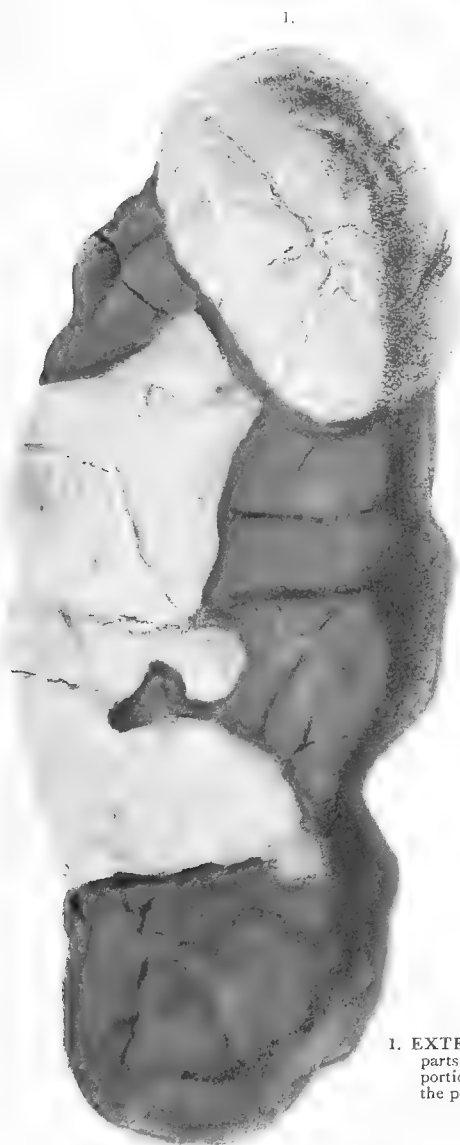
BRONCHO OR CATARRHAL PNEUMONIA, CORN-STALK DISEASE,  
TRANSIT PNEUMONIA.

Pleuro-pneumonia has been confounded with another disease—broncho-pneumonia, called by American veterinarians the "corn-stalk disease," and transit pneumonia by the Canadians, described by Billings as interstitial pneumonia, following a septicæmia induced by eating decomposing corn-stalks (Indian corn-stalks)—by the veterinary officers of the Board of Agriculture and others, and I feel warranted in relating the history of that disease as seen in this country.

Early in 1879 pleuro-pneumonia was reputed to have been found amongst American cattle landed at Liverpool, and the



2. INTERNAL ASPECT OF No. 1.  
Bronchial Tubes filled with catarrhal  
products. Lung tissue engorged.



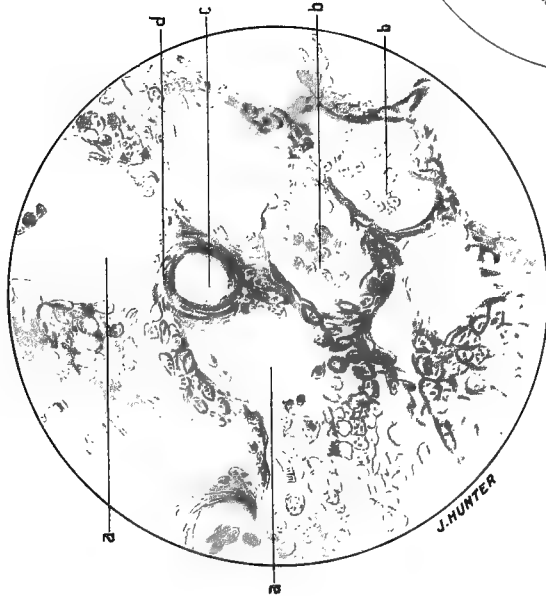
1. EXTERNAL ASPECT, showing red  
parts, collapse of lobules, and at lighter  
portions some degree of emphysema of  
the pervious lobules.

**BRONCHO-PNEUMONIA, OR SO-CALLED AMERICAN  
CONTAGIOUS PLEURO-PNEUMONIA.**

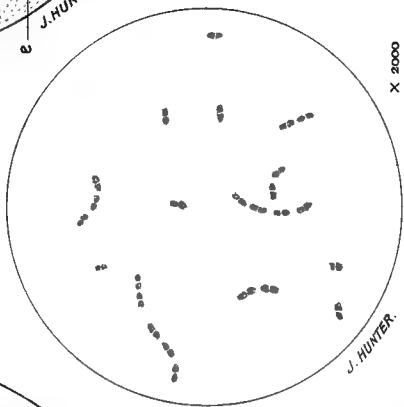




# PLATE VIII.



BACTERIA  
OF BRONCHO-PNEUMONIA,



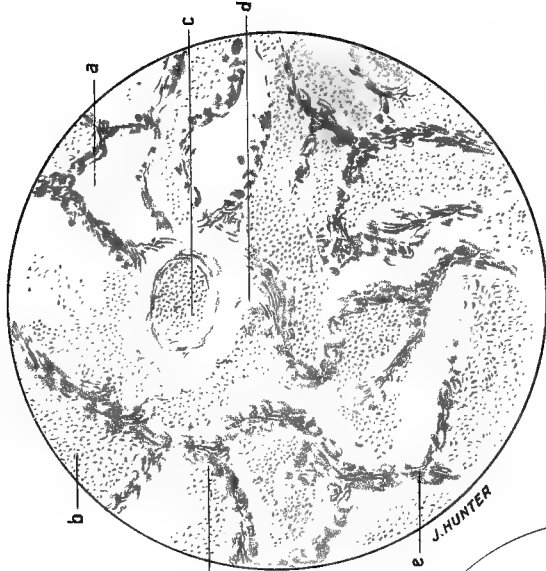
Average width =  $\frac{1}{1000}$  of an inch.

## BRONCHO-PNEUMONIA.

(Stained with *Picro-Carmine*. X 300.)

From Lung from Canadian Ox, killed at Lindores, Fife, October 1892, and stated by Officers of Board of Agriculture to be *Pleuro-Pneumonia Contagiosa*.

- a a* Air cells free from deposit, with their epithelial cells more or less desquamated.
- b b* Masses of shed and proliferated epithelium occupying several of the air cells.
- c* Blood-vessel with its lining entire—no deposit.
- d* Fibrous walls of above rather condensed than opened out. Especially surrounding the bronchi, but also elsewhere in the lung, the connective tissue shows changes due to interstitial inflammation.



## PLEURO-PNEUMONIA CONTAGIOSA.

(Stained with *Picro-Carmine*. X 300.)

- a* Air cells in some instances free from fibrinous deposit—epithelium normal.
- b* Most of the air cells filled with the fibrinous deposit of a non-cellular and granular character.
- c* Blood-vessel filled with similar deposit.
- d* Fibrous walls of above opened out and infiltrated with some deposit.
- e e* Epithelium and air cells seen to be normal where it is not optically covered by the granular deposit.

To face page 189.

shippers there, in order to satisfy themselves of the correctness of the diagnosis of the veterinary advisers of the Privy Council, and who now hold the same position on the Board of Agriculture, requested Professors M'Call, Walley, and myself to visit Liverpool and examine the cattle, and watch the *post-mortem* results; and here commenced the dispute, as, after having seen a few diseased lungs, I maintained that the malady was not pleuro-pneumonia, but a catarrhal or broncho-pneumonia, combined in a few cases with pleurisy, but in others the pleurisy was absent. Professors M'Call and Walley agreed with the advisers of the Privy Council, and I found myself in a magnificent minority. During the spring and summer of that year several condemned lungs were sent to me from Liverpool, and after further investigation I found no reason to change my opinion.

The matter remained in abeyance so far as the profession was concerned until April 1891. It is very true we now and then heard that pleuro had been detected amongst American cattle landed in this country, but we had no means of verifying or contradicting these reports; but some two years or so ago the American Government sent inspectors to the various ports where American cattle are landed, for the purpose of investigating and reporting upon the cattle slaughtered; and, wonderful to relate, we heard no more of American pleuro, except one notable report from Dundee, until a lung of an ox was found at Deptford in April 1891, and said by Professors Brown and Duguid and Mr. Cope, and confirmed by Professors Walley and M'Fadyean, to have *pleuro-pneumonia contagiosa*, and in further support of the correctness of the diagnosis Mr. Chaplin, the Minister of Agriculture, said it was pleuro, *for he had seen the lungs*. But Dr. Wray, the American veterinary inspector at Deptford, disputed this opinion, and on 18th April brought a portion of the lungs to me.

I, along with my son, examined this portion of lung, and we both came to the conclusion that it was not pleuro-pneumonia. I was again in the minority, and nothing more would have been heard of the matter, at least so far as I am concerned, had not a remarkable coincidence occurred in Paris during the winter of last year, when it appears that MM. Redon, Godbille, and Blier, well-known veterinarians and sanitary inspectors at La Villette—the Parisian cattle market—where thousands of American cattle

were exposed for sale. On three several occasions these veterinarians affirmed that some of the animals were affected with an unusual disease of the respiratory organs. In November, 1890, in a lot of more than 400 cattle direct from Illinois and Indiana, one died, and two were very ill, the symptoms leading to the suspicion that they were affected with contagious pleuro-pneumonia.

"The sick animals were killed, and the lungs examined by Godbille, who found no pleurisy, but such a suspicious appearance that, considering the importance of the matter, the two



*Photo-Micrograph by W. Forgan.*

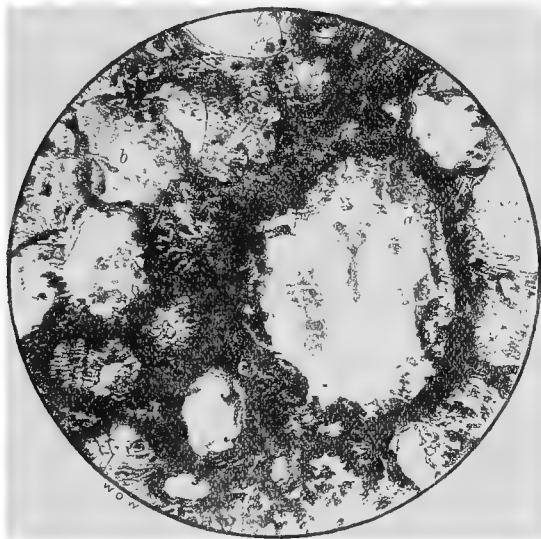
FIG. 14.—Pure Pneumonia as seen in Pleuro-Pneumonia.

- a.* Healthy epithelium of bronchus.
- b.* Fibrinous plug.
- c.* Air vesicles containing croupous—fibrinous—exudate.

sets of lungs were sent to Alfort, to be submitted to M. Nocard, one of the most illustrious of veterinarians and pathologists, and he reported that at the first glance a section of the hepatised tissue presented the appearance of a recent lesion of *pleuro-pneumonia contagiosa*. The tissue was dense, compact, friable, the colour varied from bright red and deep brown to almost black, whilst the lobules were isolated from one another by thick septa, infiltrated with a considerable quantity of yellow limpid fluid, etc."—See *Veterinary Journal*, October, 1891.



These appearances were startling, and if the investigator had been a less eminent man than he is, he might have made his conclusions from these naked eye appearances, and said it was contagious pleuro; but there were great international and commercial considerations in question, as well as the scientific bearing of the subject; and Nocard did what our own Government officials ought to have done, if not in 1879, at least in 1891. He made further investigations, and arrived at the



*Photo-Micrograph by W. Forgan.*

FIG. 15.—Broncho-Pneumonia as seen in "Corn-Stalk" Disease, from American ox slaughtered at Deptford, 14th April, 1891, and referred to in text.

- a.* Bronchial walls, epithelium desquamating and shed.
- b.* Catarrhal products in tubes and vesicles.
- c.* Increased fibrous tissue—fibrosis.

conclusion that he had other than pleuro to deal with. He found, in the first place, that the infiltration of the connective tissue was less abundant, the serum less albuminous; that the tissue of the lobule in the thickened girdle of connective tissue had not the uniformity of tint and consistence that characterise pleuro-pneumonia lesion; that it was harder, more manifestly hepatised in the centre than at its periphery—the invasion being from the bronchial, and not from the perilobular tissue. "In pleuro"

it is the opposite, notwithstanding what Drs. Yeo and Woodhead have stated, and which statements have been partly accepted by Professor M'Fadyean.

There was another important differential sign. Pressure caused a notable quantity of thick, viscid, light yellow pus—analogous to that observed in certain forms of verminous broncho-pneumonia—to issue from the bronchioles (this condition was very manifest in the Liverpool cattle in 1879); but a microscopic examination of this fluid and of the lung-pulp did not lead to the detection of ova, embryos, or worms of any kind. The bronchial mucous membrane was inflamed, thickened, corrugated, and more or less denuded of its epithelium, while the submucous connective tissue was infiltrated with yellow serum, and considerably thickened in places; but the blood-vessels contained no thrombi.

This muco-pus from the bronchioles was found to contain an abundance of short, oval, mobile bacteria, which appeared to be the only microbes present. The organisms were also found, as if in a state of pure culture, in the hepatised tissue, and more especially in the limpid serosity that distended the perilobular lymphatic sac. This single character alone sufficed to affirm that the lesion was not of a pleuro-pneumonic kind; for it is well known that the lung serum in that disease is very poor in figured elements, and that when it is collected pure from the infiltrated septa it does not usually contain any microbes.

In this instance the microbe, which existed in great numbers, belonged to the large class of ovoid bacteria of which the rounded poles fix aniline colouring matters more strongly than the centres. It is well known, as pointed out by Nocard, that the fowl cholera, the duck cholera, the septicæmia of rabbits and ferrets, the game plague, the swine plague, &c., all have analogous microbes, and which can only be distinguished from each other by the collective biological characters, and more especially by the effects of their inoculation on different kinds of animals.

Experimental inoculations were now made by M. Nocard, when he found that mice, rabbits, guinea-pigs, and pigeons, when inoculated subcutaneously with two or three drops of the serum or the culture, succumbed in less than forty-eight hours, without œdema at the point of inoculation, with intense congestion of all the viscera, but without any definite localisation.

Sheep and calves inoculated subcutaneously, or in the trachea, with a cubic centimetre of culture, of serosity, or of virulent pus, did not die; they suffered, however, from intense fever, and remained prostrate, without appetite, for some days, but soon regained their normal condition.

Intra-pulmonary inoculation was more effective, as a calf of eight months and a ram of two years, inoculated in the right lung with five drops of peritoneal pus from a guinea-pig, died in less than forty-eight hours with fibrinous pleurisy and exudative broncho-pneumonia, analogous to that observed in American cattle, the lesions being very rich in bacteria. Nocard concludes that the disease does not resemble any known in France, and considers it is a malady special to American cattle, and is inclined to think that it is the affection known in the Western States as the "corn-stalk disease," described by Gamgee, and more recently by Billings, of whose observations there is an account by Bowhill in the *Veterinary Journal* for February 1892. And the question arose as to whether the importation of this microbic broncho-pneumonia would prove a source of danger to France. M. Nocard was able, however, from his close observation of facts, and the experimental study of the conditions of contagion, to state that the malady had only very feeble contagious properties. In three very large importations in which the disease was detected, notwithstanding the considerable number of animals in each, and their long and close contact with each other, the affection did not spread, and the cases remained isolated ones. From all these facts, his opinion was there was no urgent danger, or any necessity for special measures.

Dr. Fleming, in a leader upon M. Nocard's observations (see *Veterinary Journal*, October 1891), says:—"The question that now arises on this side of the Channel is one of some moment, not only from an economical, but also from a pathological point of view. Many cargoes of American cattle have been condemned because it was found that one or two in each lot were affected with what was supposed to be the specific lung plague. United States veterinary surgeons have strenuously denied the existence of that malady among cattle, and Professor Williams on two occasions, when the matter has been referred to him, has concluded that lung plague was not present, but the lesions were those of catarrhal or broncho pneumonia; and now Nocard finds

what we are almost forced to conclude is the same disorder, examines and experiments with it, as a scientist of his position and responsibility should do, and—*mirabile dictu*—arrives at the same conclusions as Williams, and even designs it by the same name.”

Notwithstanding this, the North of England Veterinary Medical Association passed a formal resolution—“That the best methods of recognising pleuro were by the naked eye and hands, and that when these methods failed, little reliable assistance could be obtained from the examination of microscopic sections; that is to say, as far as determining it to be absolutely a case of pleuro-pneumonia was concerned.”—(*Veterinary Record*, 17th December).

At a meeting in Liverpool on 16th December 1891 a description was given by M'Fadyean of the *post mortem* appearance of pleuro, and a careful perusal of that description will at once convince any impartial reader that he labours very hard to prove the catarrhal origin of that disease. In one passage it was stated by M'Fadyean that—“The alveolar contents are by no means always fibrinous in character. In many lobules the change is distinctly a catarrhal one, air vesicles being filled with round nucleated cells, apparently the progeny of the swollen and proliferating epithelium. This change may affect the lobular bronchi also. Sometimes the catarrhal proliferation is associated with a peculiar transformation of the alveolar epithelium, that having become distinctly columnar in character. The fact that catarrhal changes are common in contagious pleuro-pneumonia has been strongly insisted on by several writers upon the subject, notably by Dr. Gerald Yeo and Dr. Woodhead.”—(*Veterinary Periodicals*.)

With regard to the conclusions of Yeo, I have already demonstrated their fallacy; and with reference to those of Woodhead, published in the *Journal of Comparative Pathology, &c.*, 1888, I wish to draw attention to a review of that article by M. Delaforge, sanitary veterinary surgeon for the Department of the Seine—*Recueil de Méd. Vétérinaire* for 15th May 1889, page 324. He says:—“In an analysis of ‘Studies on the Pathological Anatomy of Pleuro-Pneumonia,’ by Sims Woodhead, which appeared in the *Recueil* for 15th March, I read as follows: ‘The pathological process consists in an irritation of the mucous membrane of the smaller bronchi, followed by shedding of the epi-

thelium of the pulmonary vesicles and a catarrhal proliferation of their elements. The virus then penetrates from these vesicles and the smaller bronchi into the neighbouring lymphatics (peribronchial lymphatics), but not into those of the bronchi," &c. From this it appears that this author's opinion is that the initial start of the malady is in the bronchioles, and from these to the lymphatics or lymphatic spaces—the perilobular networks. This idea is radically false. Sims Woodhead mistakes the epiphenomena for the primordial phenomena—such as clinical observation shows very distinct. Every practitioner who cares to take the trouble to inform himself otherwise than through his imagination will readily perceive that at the periphery of the hepatised mass there are almost constantly met with, in an extent of 5, 10, 15, and 20 centimetres, connective tissue bands, with their ramifications distended by the exudate of pleuro-pneumonia, without any alteration, any change even in colour, in the pulmonary lobules, which are surrounded by these advanced-guard projections. When two such projections run parallel at a short distance from each other, we can clearly see, from their point of emergence from the hepatisation, the graduated effects of the compression they exercise on the imprisoned strip of lung. The congestion and density of the lobules is then marked in decreasing proportion from the base to the terminal divergence of the two infiltrated bands (or spaces); while, on the contrary, the lobules outside them and touching them remain absolutely healthy. The clinical facts, which are very common, I repeat, prove distinctly that the evolution of contagious pleuro-pneumonia takes place from the connective tissue septa (interlobular spaces) towards the pulmonary vesicles, and not—as Sims Woodhead asserts—from the bronchioles towards these septa. The obstruction of the vesicles, the catarrhal and ephithelial transformations, the blood stasis, the pulmonary hepatisation, in a word, are evidently not the initial essence here, but rather physical corollary or epiphenomena. It is in this course, by this order in succession of the pleuro-pneumonia lesions, that in a first case, for instance, the practitioner can most certainly affirm the contagious nature of the malady; for whatever may be said and written to the contrary, there is observed in the bovine species a non-specific pneumonia and pleurisy, which may or may not be coincident, and the altera-

tions in which are physically similar to those of contagious pleuro-pneumonia. I have had cases, the circumstances attending the origin of which, as well as the consecutive circumstances, left no doubt in my mind with regard to this. And apart from inoculation, which is not always practicable, I did not know until then of anything that had been published that informs us how we can differentiate between what is specific and what is not. Nevertheless, this means is, if not always constant, at least so in the majority of cases. To find it, it is only necessary to make an attentive and minute examination of all the periphery of the diseased parts; if only for a few centimetres, we find an interlobular exudation advancing into the soft rosy tissue, we may be certain that we have before us the contagious pleuro-pneumonia. It is less with the view of rectifying the above error than of establishing this indication that I request the insertion of this short note."

Further, in order to prove the catarrhal theory, M<sup>r</sup> Fadyean says that the piece of lung brought to me by Dr. Wray might have had broncho as well as pleuro-pneumonia. I do not deny the possibility of the two conditions being sometimes—but, as a matter of fact, very rarely—co-existent; but it is a remarkable thing that the very many specimens of American and Canadian lungs that I have seen since the commencement of 1879 should all have been catarrhal, and the many hundreds of pleuroed ones that I have seen in this country should have the catarrhal conditions absent, and since then I have received a portion of another lung from Dr. Wray, in which the lesions are identical with those found in the 1891 lung. Portions of this lung were sent to M. Nocard, who kindly examined them, and I now append his opinion:—

“ALFORT, 29th December 1891.

“MY DEAR COLLEAGUE—You will excuse my not having replied sooner, but I did not wish to limit myself only to an examination of the sections you sent me; for I was desirous of thoroughly studying the piece of lung you also forwarded. This study I have now made comparatively, with pieces I myself collected at the commencement of this year or end of 1890 from American cattle, the history of which I brought before the Société Centrale Vétérinaire in July last, under the title of ‘Infectious Broncho-Pneumonia.’

“I have made a great number of sections of your specimens and of my own, and have treated them by the same methods of

staining, which I have varied as much as possible. I have examined them with much care, and I believe I am now in a position to conclude:—1. The lesion which you have submitted to me is certainly not that of pleuro-pneumonia; 2. It is a bacteridian broncho-pneumonia, which, *in all probability*, is of the same nature as that I have already described. On the latter point I am not so positive as on the first, for although the distribution is very analogous, and the microbes have the same form and the same dimensions, and react in the same way to the different staining methods, you know as well as I do that we cannot, on these alone, affirm the identity of two microbes. In order to do this we must cultivate your bacteria, and then study them comparatively with mine, to see how they behave in the different culture media, and observe their pathogenic action on various animals. By doing this I shall doubtless be able to affirm or deny that we are dealing with one and the same organism. However this may be, I again repeat that it appears probable to me that you and I have studied one and the same disease.

E. NOCARD."

A statement having been made that the microbe found was an ordinary putrefactive one, I wrote to Dr. Wray, and I append his reply, with the simple observation that when the lung arrived in Edinburgh it was free from putrefaction:—

"NO. 39 BELVEDERE ROAD, UPPER NORWOOD,  
LONDON, S.E., 8th January 1892.

"W. WILLIAMS, F.R.C.V.S., &c., Principal,  
New Veterinary College, Edinburgh, Scotland.

"MY DEAR PROFESSOR—In reply to yours of the 6th instant, the section of lung that I showed you on 18th April 1891 was taken from the lungs of a bullock that was slaughtered at Deptford on the afternoon of 14th April 1891. The lungs from the said bullock were sent to the Royal Veterinary College the same afternoon. The next morning, 15th April, I inspected the lungs in company with Professor Duguid, and Doctors Coghill and Shaw, the section I took you being cut off immediately after the inspection, Professor Duguid cutting off an adjoining section at the same time. The section I had was placed in a tin box, and there kept until I arrived home the same evening, was then taken out of the box and wrapped in a linen cloth saturated with a solution of bichloride of mercury [1 to 1500], and placed on ice, where it remained until 10 P.M. of the 16th, when it was taken out of the cloth, re-saturated with the solution, then packed in a clean box, having been removed from the box twice previous to your seeing it on the morning of the 18th. I took the utmost care

to get the section to you in as fresh a state as possible. When you saw the section I remember distinctly that it was quite fresh and free from any signs of putrefaction. If I can be of any assistance to you in this case, please command me. With many happy returns of the past holiday season to you and yours.—Very respectfully,

“W. H. WRAY.”

Now, in conclusion, I have simply to say that the American disease is not pleuro, but that it agrees in every respect with what Gamgee, Billings, and Nocard have described as broncho-pneumonia, and that it is identical—but the specimens show a more chronically diseased condition—with what I discovered in both States and Canadian cattle landed in Liverpool in 1879.



## CHAPTER XVIII.

### CONTAGIOUS DISEASES—*continued*.

#### ECZEMA CONTAGIOSA.

*Synonyms*.—Eczema epizootica; epizootic aphtha; aphthous fever; foot-and-mouth disease; murrain; epidemic.

*Definition*.—A highly contagious and infectious febrile disease, associated with a vesicular eruption in the mouth, between the pedal digits, and around the coronets. In some cases the mouth only is affected, in others the feet may be the seat of the eruption, the membrane of the mouth remaining free. In milch cows it sometimes happens that a vesicular eruption occurs on the mammary gland, and within the lactiferous ducts: when such occurs, the milk, contaminated by the vesicular discharge, is rendered unfit for use, either as food for the human being or for the lower animals, as it is said to induce a vesicular eruption in the mouth, larynx, pharynx, and intestinal canal.

#### PATHOLOGY AND SYMPTOMS.

Unlike rinderpest and pleuro-pneumonia, which are almost confined to the ruminantia, contagious eczema affects cattle, sheep, pigs, dogs, poultry, and even human beings are subject to the influence of its virus. It is amongst horned cattle that we find the ravages of this disease to be most extensive, they seemingly being most susceptible to the influence of the contagious poison; sheep and goats, however, suffer very severely, and dogs, pigs, and poultry often die from it.

After a period of incubation, varying from twenty-four hours to three or four days, the invasion and progress of the disease are characterised by an elevation of temperature of from two to four

degrees ; by the formation of vesicles, varying in size from that of a sixpence to that of half-a-crown, on the tongue, inside the lips, roof of the mouth, and sometimes on the udder ; whilst smaller blisters make their appearance between the digits, and around the coronets and heels, and there is a discharge from the eyes and nose. The animal presents signs of uneasiness in the mouth, by constant movement of the lips, champing of the teeth, a flow of saliva from the mouth, and difficulty in masticating its food ; the affection of the feet being indicated by some degree of lameness in the foot or feet affected. In a short time the epithelial and cutaneous structures enclosing the vesicles are separated and thrown off in more or less rounded patches, leaving raw surfaces (see Plate VI., p. 182), which are, however, speedily re-covered by epithelium. In some instances there is an entire separation of the hoofs from the feet.

The symptoms are undoubtedly due to the presence of a contagious micrococcus in the animal economy, and are manifestations of the elimination or excretion of a virus, which evidently has some special affinity to the mucous and cutaneous structures of the several parts of the body which it invades. Eliminated by the feet and by the mouth, pastures very soon become impregnated with it, and hence we find that, owing to the great facility by which it is thus spread over a wide district of country, it appears to be the most infectious of maladies. Beyond question the virus of eczema is conveyed from field to field, from parish to parish, from county to county, by small game, vermin, and dogs ; and looking to this fact, we can account for the sometimes mysterious outbreak of the disease in places and districts into which no fresh cattle have been admitted. This frequently unaccountable appearance of the foot-and-mouth disease has led to the belief that it is of spontaneous origin ; some believing that, although originating spontaneously, it is propagated by contagion ; others that it originates in all cases independently of contagion, and that it is due to climatic or atmospheric causes, exposure, and the debilitating effects of long journeys by land or sea. It is very true that increased susceptibility to the disease is induced by any debilitating cause, but neither age, condition, management, climate, temperature, nor any common cause of disease seems to exert any modifying influence. No extremity of privation, nor the continued action of ordinary causes, is

capable of inducing it; and one reason for the indifference which has been shown in respect to its ravages is to be found in the belief in its spontaneous origin, an idea which arises out of the observations of its frequently unaccountable appearance in isolated places.—(Professor BROWN on *Mouth and Foot Complaint*.)

It was at one time believed that an animal which had once suffered from eczema contagiosa was guarded against another attack. This, however, is a fallacy; and it is now generally believed that an animal is only once attacked during a season. As a general rule this may be said to be the case, and consequently a herd which has gone through the disease early in the season—let that be the spring or autumn, the periods when, in consequence of the increased movements of cattle from place to place, the disease is most rife—is considered more valuable to the purchaser, and worth more money in the market. This rule is, however, liable to many exceptions, and it is by no means impossible for the same animal to suffer from two, three, or even four attacks during a period of a few months.

Milk from cows affected with foot-and-mouth disease acts energetically upon young animals to which it is given warm. Calves occasionally die quite suddenly after sucking cows affected with the malady; and fatal effects have followed the administration of the milk to young pigs.

Not only is the disease communicable from cattle to sheep, goats, and pigs, but it is also to children, and even grown-up people, as well as to numbers of other kinds of animals.

It is now established that the causal agent in foot-and-mouth disease is an ultra-microscopic organism. The exudate from the buccal or pedal vesicles may be filtered through a fine porcelain filter, so that the filtrate is free from all visible organisms; and yet this filtrate will produce the disease. This eliminates the possibility that any of the visible cocci or bacteria are in any way the causal organisms, and their presence there is only accidental, as would be natural in any skin-lesion. This disease then comes under the same classification as pleuro-pneumonia, rabies, South African horse-sickness, etc. The virulent filtrate obtained does not yield any growth upon cultivation in any known media, and we can only surmise that the organism, although existent, is invisible.

Cows, when suffering from the worst form of disease, lose

nearly all their milk; but when the attack is mild in character, the decrease will not be more than one-third of the usual yield. The average loss in a large dairy while the disease is going through the sheds will vary from one-third to two-thirds, according to the number of severe cases. As all the milk obtained is mixed, the worst milk will be to some extent modified by the addition of that which is less highly charged with morbid elements, and the whole is further diluted by the addition of water, which, judging from some specimens obtained from an establishment where the disease was known to exist among the cows, is sometimes added to the extent of 40 per cent. Boiling the milk has been recommended for the purpose of preventing or lessening its injurious action.

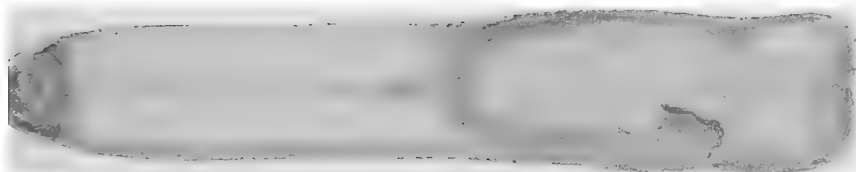
In ordinary cases of eczema the symptoms are not very severe, and provided the feet are not seriously implicated, the animals recover in about a week from the first manifestation of the febrile symptoms. In many instances, particularly if the weather be cold and the cattle exposed, a hoarse tracheal cough is present, with much discharge from the trachea and bronchial tubes, whilst in aggravated cases the mucous membrane of the intestinal canal is very seriously involved, and when vesicles appear at the anus, it is fair to presume that vesication exists along the whole alimentary track; and *post-mortem* examinations of such reveal the presence of much inflammation of the intestinal mucous membrane, patches of denudation here and there, more especially in the fourth stomach, giving the disease very much of the characteristic appearance found in cattle plague, to which disease it is to some extent allied.

It is when the feet are much affected that the loss to the stock-owner becomes great, for not only does the animal lose condition from the disease, but also from inability to roam about its pasture in search of food. Such cases should at once be comfortably housed, provided with proper aliment, and have the feet properly treated.

In sheep, goats, and pigs the feet are the principal parts affected, the vesicles occurring between the digits and around the coronets.

During the summer of 1872 eczema contagiosa assumed a very malignant character, especially in some herds of Iceland

PLATE IX.



FOOT AND MOUTH DISEASE. .

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EXCORIATIONS ON TONGUE.

*(From a Model in Museum of New Veterinary College.)*



cattle brought to Leith and Edinburgh, the ordinary symptoms being complicated with swelling of the legs and other parts of the body, the swelling when opened discharging a very foetid fluid. In all there was great prostration of strength, and many cases terminated fatally.

The loss from foot-and-mouth disease to the dairyman, from the diminished secretion of milk, and to the owner of cattle nearly fit for the butcher, is very great. I do not think £4 a head too high an average; but when it is confined to lean or store cattle, provided the attack is mild, the loss of condition is quite ephemeral, and can scarcely be accounted a loss at all, for such animals appear to make flesh much faster after than prior to an attack of the disease.

In India, where foot-and-mouth disease is common, it is as a rule a comparatively harmless affection, and working bullocks often continue to work without any apparent difficulty. If, however, these animals are severely tried, as happens upon a campaign amongst draught animals, the affection is liable to assume a severe or even dangerous type, and sequelæ such as extensive gangrene of the foot may supervene. If an outbreak of a contagious disease such as rinderpest should affect such animals, the death-rate will assume alarming proportions. In that country, amongst the cattle of the plains, rinderpest is usually fatal to about 33 per cent. of the animals, but if foot-and-mouth disease be present the death-rate will be 90 to 100 per cent.

As sequelæ to eczema contagiosa, the following alterations of structure have been observed—namely, swelling of the joints, open joints, with sloughing of interdigital substance, gangrene of a whole foot, marasmus, and disease of the bones.

#### TREATMENT OF ECZEMA CONTAGIOSA.

Mild cases require little medical treatment, foot-and-mouth disease being one of that kind which, running through a definite course, terminates in recovery. Whilst the mouth is very sore, all that the animal requires is a plentiful supply of cold water; if the fever be high, the water may contain an ounce of nitrate of potash dissolved in each bucketful. As a rule, however, even this is unnecessary. The food should

be soft and easy of mastication—bran mash, pulped roots, or grass. The feet must be looked to, and if suppuration be detected, with separation of some parts of the horn, the detached horn is to be removed, and the exposed sensitive surfaces dressed with a mild astringent, such as a solution of acetate of lead, containing a small modicum of carbolic acid. What I usually prescribe is an ounce of the concentrated solution of pure carbolic acid—one part acid to sixteen hot water—to twelve of the white lotion. Pledgets of tow dipped in this lotion are to be bound to the foot by a bandage. This simple expedient, so generally neglected, will often prevent a lameness of many weeks' or even months' duration; for suppuration, once established to any great extent in the sensitive structures of the foot, causes violent pain, increases the inflammation, and induces the process of sloughing. Some writers recommend a variety of remedies both to the mouth and feet. My experience leads me to the conclusion that, except carefully washing the feet, the less medical interference the better.

Now and then cases will be marked by extreme prostration of strength, entire loss of appetite, inability to stand from weakness as well as from the lameness; such ought to be more actively treated, and stimulants, such as whisky, brandy, or spirits of nitrous ether, administered internally, and the feet washed repeatedly with cold water, and kept cool with wet cloths fastened around them. In all instances, bleeding, purging, and other depleting measures are most injurious. The bowels, perhaps constipated at first, become loose as the disease advances; but this condition is not to be interfered with, for the diarrhoea may be looked upon as one result of the *vis medicatrix naturæ*, by which the morbid material is expelled from the system. Extensive sloughing of the structures of the feet is best treated by more powerful antiseptics—solutions of chloride of zinc, or strong solutions of carbolic acid, say one part of crude acid to six or eight of oil.

Sheep lose flesh very rapidly whilst suffering from foot-and-mouth disease. They suffer more in the feet than cattle, and consequently require more careful vigilance. It is a good plan to cause the affected flock to walk through a shallow trough, containing the above-named weak astringent and antiseptic solution, once or twice a day. And finally both cattle and



sheep ought to be washed before they are allowed to be driven on a public road or exposed in a market, if they have recently recovered from this disease. It may be difficult to do this with large herds of young cattle, and its compulsion would be considered a hardship, but of two evils it is incomparably the lesser.

Pigs are frequently affected by receiving the milk from cows suffering from the affections, and have very well-marked lesions around the coronets. In these animals the disease is of a comparatively mild form, and they soon recover under ordinary treatment.

Children contract the disease by drinking infected milk, and suffer from sore throat, aphtha or thrush, and often an eruption of vesicles between the fingers.

## CHAPTER XIX.

### CONTAGIOUS DISEASES—*continued*.

#### INFECTIOUS ABORTION.

ABORTION in cows is often seen as an epizootic, both in this and other countries, and its pathology has been profoundly studied in France and Switzerland, more particularly by Nocard, and its clinical aspect by Strebel, Galtier, de Poncius, and Org; by Lavat, who has witnessed it in sheep; by Biot, who considers it is an infectious inflammation of the serous lining of the uterus; and by Herr G. Sand, Copenhagen, and Professor Bang. In this country, M'Fadyean, Woodhead, and Aitken endeavoured to throw light upon its pathology. Their experiments were repetitions of those of Nocard, and lead to no fresh conclusions; but a very valuable paper was read upon the subject by Mr. Harry Olver, F.R.C.V.S., Tamworth, at the meeting of the National Veterinary Association at Birmingham, 1895.

Mr. Olver, who seems to have had great practical experience, rightly describes the disease under two heads—namely, non-contagious and contagious abortion—and states that abortion occurs at from the third to the seventh or eighth month. He also states that sporadic abortion is due to many causes, such as cold, wet, fright, injuries, food, water (when drunk icy cold); but he traverses the opinion of C. Stephenson and others, that stagnant water contaminated by sewage has any appreciable effect,<sup>1</sup> though since then it has been fully proved that in-calf cows which drink water contaminated with sewage are likely to suffer from its ill-effects and to abort.

Contagious abortion rarely occurs in the mare, ewe, or goat, and still more rarely as an epizootic in this country, but

<sup>1</sup> See Transactions of National Veterinary Association, 1895.

cases are recorded of undoubted outbreaks in America, France, Australia, &c.

The disease occurs at all periods of gestation after the third month, and in the same animal at a later stage in each succeeding pregnancy, until at length the foetus is carried its full term, if the mother has not become sterile. The calf is generally dead at birth, but in some instances it is born alive; it is, however, generally in bad health, emits a peculiar lowing, and after the third day is attacked with diarrhoea, which rapidly terminates fatally.

It is true that there is often great excitement amongst the cows in a byre during and after abortion by one of them, and that there is also often a peculiar odour emitted by both mother and offspring. It has been attributed to ergotism, mouldy food, cold, contusions, unwholesome drinks, &c. All these may be predisposing causes, but it is now fairly demonstrated by Nocard and Bang that the virus exists in the discharge from the mother, and in the uterine fluids, and that by the introduction of these fluids from an aborted cow into the vagina of healthy ones abortion will be induced in from nine to twenty days.—(BRAUER.) Lehnert thus produced it in twelve to twenty days, and Trincherra obtained a purulent vaginal catarrh and abortion in from nine to nineteen days. Inoculation matter obtained by scraping the surface of the chorion of an aborted cow produced a like result. The transmission from cow to cow is most probably by immediate contact of the material with the genitals of cows in the same byre, the discharges being conveyed by the groop or drain which is immediately behind the cows in all byres.

It can easily be understood how the virus thus conveyed contaminates the tails and hind extremities of the cows when recumbent, and how easily natural inoculation is thus effected by the movements of the tainted tails, more especially if the drain leads from the aborted to the healthy ones.

Nocard states that abortion takes its origin in diverse germs met with in the uterus of aborted cows, but which are never found in healthy ones. These germs are also present in the amniotic fluid in the alimentary canal of aborted calves, as well as in the medulla oblongata of those which during life give utterance to the peculiar lowing sound already referred to.

Nocard states—"The autopsy of diseased animals does not reveal any alteration of the thoracic or abdominal viscera, with the exception of the uterus. The peritoneal serous membrane is normal, incision of the uterus reveals the presence between the mucous membrane and the chorion of a fibrinous mucopurulent matter, which is more or less abundant and often acid, in which the microscope shows epithelial cells, leucocytes, a large number of isolated micrococci, which are generated or associated in short chains of three, four, or five sections, and a few short bacilli, which are isolated or associated in pairs; in the cotyledonous liquid the bacilli are predominant; in the product obtained by scraping the uterine mucous membrane both micro-organisms exist in almost equal numbers. The amniotic fluid also contains them. In the aborted foetus the intestinal mucous membrane is the seat of abundant epithelial desquamation; its tissue seems infiltrated with various microbes, which also exist in abundance in the contents of the intestines. These micro-organisms, which are contained in the digestive tube, give an explanation of the diarrhoea by which the calves which have been aborted in an advanced state of gestation are affected in two or three days after birth. Micrococci identical with those found in the amniotic fluid are also found in the medulla oblongata in those calves which continually bellow during the days preceding death." Nocard arrives at the following conclusions:—

1. In aborted cows, even in primipara, there exists in the uterus, between the mucous and the foetal membranes, especially in the cotyledonous crypts, several micro-organisms which cannot be found in newly calved cows, which belong to a locality where abortion does not exist.

2. These micro-organisms do not seem to exercise any noxious action upon the uterine mucous membranes of the mother, either during the period of gestation, which must be interrupted suddenly, or even after abortion.

3. Repeated abortion in the same subject can easily be explained, if we admit the pathological influence of the microbe, by the persistency of the latter in the uterine cavity till the time when it is able to exercise its action on a new foetus or on its envelopes.

4. Also cases of sterility which are consecutive to abortion can be explained by the acid reaction of the uterine mucus, in which the microbes thrive, the spermatozoa not being able to preserve their vital properties except in alkaline elements.

5. Epizootic abortion seems indeed to be a microbic disease of the foetus and its envelopes, but one which does not affect the mother.—(REVBET, 1889.)

This form of abortion, while prevalent in certain districts, is unknown in others, but it may be conveyed from one district to another by the introduction of cattle from an infected area. Upon this point a mass of evidence is adduced by Bang,<sup>1</sup> Royal Veterinary College, Copenhagen, who hints at the conclusion that abortion, independently of its isolated sporadic occurrence in widely separated districts, occurs in an epizootic or enzootic but non-contagious form from accidental causes, such as musty food, bad or cold water, insanitary surroundings, stall feeding, and in-and-in breeding.

In addition to the evidence as to its spread by the introduction of affected cows, proof is brought forward establishing the fact that the disease is also introduced by using bulls from an infected herd, and instances are recorded where bulls have been known to suffer from severe preputial inflammation after having served diseased cows. It is also stated that the vaginal mucous membrane is reddened, permeated throughout with cord-like swellings, and showing numerous small reddish-yellow or reddish-brown nodules. There are also small eruptions about the root of the tail, anus, and vulva. These symptoms, however, are not common, but there is often a chronic purulent discharge from the vulva.

As already stated, a first abortion occurs earlier than later ones, often at the third or fourth month, whilst the period is gradually extended in later ones, and the explanation seems to be that the microbes gradually lose their virulence, until finally the animal has immunity. This fact is very important, and contradicts the generally adopted method of eradicating the disease, namely, that of disposing of aborted cattle and replacing them with fresh ones, which in their turn become affected and maintain the continuance of the disease in a herd; whereas, if aborted cattle are kept, only those proving sterile being disposed

<sup>1</sup> See Infectious Abortion, by Jno. A. W. Dollar,—*Veterinarian*. June 1893.

of, they become immune, and the disease dies out in from two to four years, as it gradually loses its virulence, appears at a later period of gestation, and attacks fewer animals.

The best and cheapest method of disinfection is to thoroughly cleanse the byres by washing and scrubbing, and then brush them over with a hot 1 per cent. solution of chloride of lime. Everything should be thoroughly cleansed twice a year, the stalls, walls, and floors once a week, and the drains daily rinsed with the chloride of lime solution. Animals showing signs of abortion should be at once isolated and provided with separate attendants.

When the foetal membranes are retained more than twelve hours they should, as far as possible, be removed by hand, and, together with the dead foetus, destroyed by fire or buried in quicklime. The uterus should be washed out with 1 per cent. creoline, or 5 per cent. lysol solution, and this should be continued weekly or oftener as long as vaginal discharges continue (usually two or three months after calving), and the cows not put to bull until their entire discontinuance. This both prevents the spread of the disease, and allows the majority of the animals to become pregnant.

In the apparently healthy cows and heifers the vagina should be carefully washed out before they are allowed to enter the cow-shed, and afterwards the vulva, tail, and surrounding parts washed daily with the same solution, and the sheaths of infected bulls should be similarly washed out.

Or Nocard's recommendations may be substituted. They are as follows :—

(1.) The floors, &c. are scraped, thoroughly cleansed, and sprinkled with a solution of sulphate of copper.

(2.) Each week from the date of conception the vagina should be thoroughly injected by means of a large syringe with the following tepid solution :—

Distilled water,	.	.	.	.	.	Twenty quarts.
36 per cent. alcohol and glycerine, aa.	3	ounces,				℥iii.
Bichloride mercury,	2½	drams,	.	.		℥ii.ss.

The vulva, anus, and surrounding parts of all the pregnant cows should be carefully sponged with the same solution.

After removal of the foetal membranes of an aborted cow, the

uterus should be irrigated, by means of a long tube introduced to the bottom of its cavity, with eight or ten quarts of the solution (tepid), but containing only half of the sublimate.

Aborted foetuses and foetal membranes should be destroyed by fire or boiling water.

In the cow abortion generally takes place from the third to the seventh month; in the mare from the fourth to the ninth; and the act will be indicated by redness of the vaginal mucous membrane, on which pimply eruptions are often seen; there is a discharge from the vulva of a reddish fluid, and a diminished

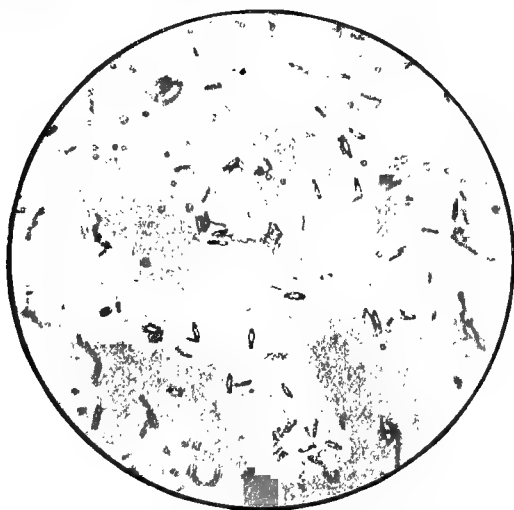


FIG. 16.—Bang's Organism and Spores of Contagious Abortion.

secretion of milk, which becomes thick, simulating colostrum. These signs may continue for three days, when the foetus is easily expelled, leaving no other symptom than an increased vaginal discharge.

The internal administration of half-ounce doses of crude carbolic acid, given three times a week in a bran mash for about a fortnight, and then discontinued for a fortnight, and again recommenced for a fortnight, and so on until parturition occurs, will enable an infected animal to carry its calf the full period and at the same time be cured of the disease.

The recent researches of Bang have thrown a great deal

of light upon this subject. He has clearly demonstrated that the disease is due to a specific organism. The means of contagion are usually by the bull, and this is accomplished by the peculiar characteristic of the organism. The effects upon the contaminated bull or cow are practically nil either locally or systematically, and it is this fact that constitutes the greatest danger in its dissemination, as there may be no physical signs that a bull is infected, and yet this animal may be the means of contaminating every cow that he serves. The effects upon the cow are also nil until she conceives, and and even then, beyond causing abortion at variable periods, generally at the third or seventh month, there may be no systemic disturbance. It is seen, therefore, that the organism may lie dormant upon the penis of the bull or in the vagina or uterus of a cow for an unlimited period, and in the latter eventually cause abortion. It is said that cows tend to become immune; there is less chance of abortion the second time; and after the third time the animal will be altogether immune.

#### PROPHYLAXIS.

The means of prevention are to cease using any suspected bulls; cows that have already aborted should not be taken to a clean bull, and, if feasible, fattened and sold to the butcher. Thorough disinfection and cleanliness are essential. Attempts have been made to provide a protective vaccine, and the matter is still being investigated. The results, however, have so far not been very successful, and it would seem that, at any rate in England, it is hardly a necessary method. The organism is difficult to grow, and it is doubtful if vaccination will have the desired effect. The medium used in the preparation of the vaccine is serum broth.



## CHAPTER XX.

### CONTAGIOUS DISEASES—*continued.*

#### VARIOLA VACCINA (COW-POX).

*Synonyms.*—*Vaccine* (F.); *Kuhpocken* (G.)

*Definition.*—A contagious, febrile, and eruptive disease, known in most parts of the globe, resulting from the presence of a specific fixed virus, which is reproduced and multiplied in the animal body during the course of the malady. After a period of latency of from six to nine days, the contagium causes the development of an eruption on the surface of the mammary gland, which eruption passes through the stages of pimple, vesicle, pustule, and scab. The disease runs a definite and mild course, and destroys the susceptibility of the affected animal to another attack; but in hot countries it sometimes assumes a diffuse and severe character.

#### PATHOLOGY AND SYMPTOMS.

Cow-pox has been described by high medical authorities as a malignant disease which destroyed cattle almost as extensively as small-pox did the human race. This view of the malignancy of cow-pox is evidently the result of a pathological error, which led observers to confuse the cow-pox with cattle plague.

Jenner believed in the identity of cow-pox and small-pox, and that both had a common origin in the "grease" of the horse. Jenner did not, however, perform any inoculations of cattle with the lymph of human small-pox. The first successful experiment of inoculating the cow with human small-pox was performed at the Berlin Veterinary College in 1801; the efforts previous to that period having been unsatisfactory. In 1807 Gassner of

Günsburgh inoculated eleven cows with small-pox matter, the result of which was the production upon one of them of vesicles having all the character of vaccine vesicles, and from which a stock of genuine lymph was obtained. In 1830 Dr. Sonderland of Barmen infected cows with the disease by enveloping them in blankets taken from the bed of a patient who had died of small-pox. Various other experiments have been made to this end by Mr. Ceely of Aylesbury, Mr. Badcock of Brighton, and by Mr. Macpherson in India. Mr. Ceely, in the tenth volume of the Transactions of the Provincial Medical and Surgical Association, gives a most interesting history of the communication of the disease to cattle from man without inoculation. "At the village of Oakley, about sixteen miles from the town of Aylesbury, small-pox had been epidemic from June to October 1840. Two cottages, in which three persons resided during their illness, were situated on each side of a long narrow meadow, comprising scarcely two acres of pasture land. One of these three patients, though thickly covered with pustules of small-pox, was not confined to her bed after the full development of the eruption, but frequently crossed the meadow to visit the other patients, a woman and a child, the former of whom was in great danger from the confluent malignant form of the disease, and died. According to custom she was buried the same evening; but the intercourse between the cottages across the meadow was still continued. On the day following the death, the wearing apparel of the deceased, the bed-clothes and bedding of both patients, were exposed for purification on the hedges bounding the meadow, the chaff of the child's bed was thrown into the ditch, and the flock of the deceased woman's bed was strewed about the grass over the meadow, where it was exposed and turned every night and for several hours during the day. This purification of the clothes continued for eleven days. At that time eight milch cows and two young heifers were turned into the meadow to graze. They entered it every morning for this purpose, and were driven from it every afternoon. Whenever the cows quitted the meadow the infected articles were again exposed on the hedges, and the flock of the bed was spread out on the grass and repeatedly turned. These things remained till the morning, when the cows were readmitted, and the contaminated articles were supposed to be withdrawn. It appears,

however, that the removal of the infected articles was not always accomplished so punctually as had been enjoined, so that, on one occasion at least, the cows were seen in the midst of them, and licking up the flock of the bed which lay on the grass. These cows were in perfect health when first put out to graze in this meadow; but in twelve or fourteen days five (out of the eight) milch cows appeared to have heat and tenderness of the teats. The teats became swollen, and small, hard pimples could be distinctly felt upon them as if imbedded in the skin. These pimples daily increased in magnitude and tenderness, and in a week or ten days rose into *blisters*—vesicles—passing into brown or blackish scabs. When the teats were in this condition and very tender, constitutional symptoms of ill health became developed. Sudden loss of milk, drivelling of saliva from the mouth, frequent inflation and retraction of the cheeks, staring of the coat, tucking up of the flanks, sticking up of the back," and rapid loss of flesh, were the appearances observed. By the middle of the third week the pustules were mature, "and the crusts and loose cuticle began to be detached." One interesting fact was brought out in the course of this outbreak, namely, that heifers may be affected with variola as well as milking cows, an occurrence which up to this period was supposed to be impossible. The reason for this is obvious, heifers before calving, bulls, and oxen not being subjected to the same chances of accidental inoculation as milking cows.

The interesting papers on this subject by Mr. Ceely, published in the eighth and tenth volumes of the Transactions of the Provincial Medical and Surgical Association, and the Reports of the Vaccination Section of the British Medical Association, contain many important facts bearing upon the identity of variola vaccina and human small-pox, and are well worth the careful study of every veterinary surgeon. But whilst these experiments seem so clearly to prove the identity of the variola of man with that of the cow, those of Chauveau point to an opposite conclusion. He performed numerous inoculations with small-pox lymph on the cow, which induced papular pustules, having no analogy with those of cow-pox. The lymph from those pustules implanted on man produced small-pox and not cow-pox; and it is stated in the *Boston Medical Journal* for 1860 that during that year Martin inoculated some variolous

matter, taken from a pock upon the body of a man who had died of small-pox, into a cow's udder, and subsequently vaccinated some fifty persons with the matter derived from the cow. Most of those so inoculated were attacked, not with vaccina but with variola (small-pox), and three died. It is difficult to reconcile such a wide difference in the results of experiments seemingly carefully performed, but I am not prepared to admit that the experiments of Chauveau and those of Martin have satisfactorily proved that those of others were wrong. In 1836 Dr. Basil Thiele of Kasan, in South Russia, inoculated some cows on the udder with the virus of human small-pox, the result being the production of vesicles bearing all the characteristics of the true vaccine vesicle. The lymph so produced from the variolation of the cows continued to retain the specific properties of variola vaccina throughout seventy-five successive transmissions to the human body; and it is not stated that it had lost its specificity after so many transmissions.

In the face of such conclusive evidence, I think we are justified in concluding that in the experiments made by Chauveau, the virus, granting that it was properly selected, had not undergone that alteration during its transmission through the bodies of the inoculated animals which modifies and mitigates its virulence under ordinary circumstances, and that the virus selected by Martin from a fatal case of small-pox was not proper for an experiment where human life was at stake.

Many experiments might be quoted (Ceely, Thiele, and others) to prove that great uncertainty and difficulty often attend the attempts to transfer the virus of variola from one animal to another; that when variolous disease affects the lower animals in a malignant form, it is capable of producing by inoculation a disease of similar severity in man, and that marked improvement sometimes takes place in the energy and in the quality of the virus by subsequent removes or inoculations in animals of the same kind, after the variolous virus had been successfully implanted in one of them; it seems to become less acrid, less virulent, and to acquire increased specific activity; capable of inducing more pronounced and perfect development of vesicles, with milder and less dangerous general symptoms.

Variola vaccina has been transmitted back to the cow from

man (retro-vaccination). By this process good human lymph loses some of its activity, for, according to Mr. Ceely, the phenomena in man, after vaccination with this retransmitted lymph, appears later, smaller vesicles are produced, but ultimately, after successive reinoculations on man, it regains its activity.

The disease vulgarly termed "grease" in the horse, believed by Jenner to be the origin of variola, has nothing to do with small-pox or cow-pox; but it is placed beyond a doubt that the horse is subject to a true equine pox, and that it is transmissible to the cow by inoculation and cohabitation. It is stated that the equine pox occurs as an epizootic when small-pox is epidemic. When small-pox was raging in Edinburgh in 1872, I saw one case of what I considered genuine equine pox. The animal (a mare) presented signs of fever, had a slight cough, loss of appetite, with costive bowels for eight or ten days before any skin disease was observed. She was treated for a common cold, with fever; but at the end of the time mentioned small pimples were observed on various parts of her body, more particularly about the shoulders and back. At first these pimples were very numerous, small, and pointed; the great majority of them, however, withered, and a small scab fell off, leaving the skin bare of hair. About a dozen on each side of the shoulders and upon the back increased in size, became vesicular, and afterwards pustular. These eruptions had very much of the character of those seen in cow-pox, namely, pimples increasing in size from a point to that of a horse-bean, becoming vesicles in three or four days, and then pustules, depressed in their centres. In about ten days, or eighteen after the first manifestation of illness, dark, thick, solid scabs formed on the sites of the pustules. These scabs did not all fall off until fully a month had elapsed, and the hair was not completely restored in two months afterwards, when I lost sight of her. Jenner, Saccho, and others, have used equine lymph for the purpose of vaccination, and human small-pox has been transmitted through the horse to the cow, and so to the child in the form of cow-pox.

It has not been determined whether the small-pox poison originated in man, the cow, or the horse; whether man had the disease communicated to him from the lower animals, or whether horses and cows had it from man. The origin will most probably for ever remain a mystery, and we must be content with the

knowledge that, through the keen penetration of Jenner, the disease directly communicated by vaccination from the lower to the higher animal has proved a blessing to millions of the human race.

There are many forms of eruption in the udder of the cow, which are sometimes confounded with those of variola; but those of variola have certain characteristics which distinguish them from all other, or so-called spurious, forms of the disease. The local symptoms of the true variola are heat, swelling, and tenderness of the teats for three or four days, followed by irregular pimply hardness of the skin, more particularly about the base of the teats. The pimples may sometimes be felt in five days after communication; they assume a red hue when about the size of a pea, are very painful and hard, gradually increase in size, and in other three or four days attain that of a horse-bean, assuming a circular form on the udder, and an oblong on the teats. They rise in the centre, become more or less pointed (acuminated), containing at first a clear, and ultimately a turbid fluid. If the vesicles are broken, troublesome sores supervene, the discharge from which will communicate the disease to the milker, if he is not already protected by previous vaccination. The vesicles, if not broken, become depressed in their centres, and have an indurated margin, around the circumference of which the skin denotes active inflammation by a circle of redness, acquiring their maximum size about the tenth day, and are then pustular; and as the pustules dry, dark brown or black solid scabs or crusts form upon the surfaces. Some of these scabs may be seen semi-detached, others entirely so, exposing a raw surface, with a slight central slough. Vesicles, pustules, and scabs may be witnessed on the same teat at one and the same time, indicating the formation of new crops of vesicles at different periods. The crusts, if left undisturbed, gradually become thicker, darker, and more compact until about the fourteenth day, and spontaneously separate about the end of three weeks, leaving shallow, smooth, oval, or circular pits of a pale rose colour, with some traces of surrounding induration.

The depression in the pocks is accounted for in various ways. Dr. Petzholdt thinks it is caused by the ducts of the cutaneous glands, which are ruptured as the pustules fill with pus and mature, but which, in the early period of the eruption, bind

down the cuticle to the cutaneous glands, thus producing the pit or umbilicus.

Dr. Gustav. Simon differs from the above opinion, and says that "variola pustules are not always constituted alike. In many cases, where a central depression clearly existed, the epidermis was entirely raised from the subjacent cutis; and only at the spot corresponding with the umbilicus were both membranes united by a thin, whitish cord, which, as the microscope evidently showed, was a hair sac. The presence of a hair sac is not, however, essential to the production of an umbilicus, and a remarkable peculiarity, first noticed by Rayer, characterises the variolous vesicle in those parts of the body where neither hair sacs nor sebaceous follicles exist. Rayer found, on removing the epidermic cap of the vesicle, and wiping off the fluid collected beneath, that a little elevation existed in the centre of the denuded corium, whilst the circumference was visibly depressed below the level of the neighbouring healthy cutis. These elevations were found by Dr. Simon, who examined them microscopically, to consist of a file of papillæ of normal or nearly normal size and condition, while the depressed margin is paved with papillæ bent down or flattened. The explanation is simple. At the centre of the vesicle an organic connection (from some yet unexplained cause) exists between the cutis and cuticle, and at this point no fluid is effused; but around this centre, exudation of fluid occurs without impediment, and tends to force the cutis and cuticle asunder."—(*British and Foreign Medico-Chirurgical Review*, vol. iii.)

About the ninth day of the vaccine disease a purely vesicular eruption makes its appearance upon the udder. Within twenty-four hours the vesicles contain a pellucid serous fluid. On the following day the fluid becomes turbid, the cuticle collapses or bursts, and a thin, brittle, flimsy crust forms, and speedily falls off. Successive crops continue to form and dessicate for three or four weeks.

It is difficult to trace the origin of an outbreak of variola vaccina in a herd of cattle. Outbreaks of it seem to follow similar inexplicable paths to those which characterise other contagious diseases. Cases spring up now and then which appear to be solitary, and cannot be traced to a cause. At other times it seems to be epizootic, and prevails at several farms at the same

time. Unlike, however, the other contagious diseases of horned cattle, and unlike variola ovina, it is a disease which seldom or never proves fatal in this country. In some instances, the constitutional disturbance is rather severe, the mucous membranes being particularly prone to participate; the mouth becomes sore, and there is drivelling of saliva.

In hot climates the symptoms are succeeded by abdominal pains, profuse diarrhoea, rapid wasting of flesh, and sometimes death; but in temperate climes the general symptoms are slight diminution in the secretion of milk, with but little or no loss of appetite.

The organism or actual cause of cow-pox is another of those which must be classed among the ultra-microscopic. Various observers have described the cause as being a coccus, but in all cases this has been demonstrated as an accidental contamination, as is in most diseases with a cutaneous lesion, such as scarlet fever, measles, &c. Cocci, diplococci, and even streptococci are constant inhabitants of the skin, and if any lesion occur, they will naturally be found easily, yet although not the actual cause of the disease, they will cause complications and even grave sequelæ from the pustular condition they set up and the danger there is of absorption, resulting probably in septicæmia.

By filtering the vesicular exudate, these contaminating organisms may be eliminated, but still the filtrate is infective, showing that it contains *materies morbi* which are infinitely smaller than coccoid bacteria—in fact, so small as to be invisible with the highest powers of the microscope yet invented. What this material is we are as yet ignorant. Pfeiffer and others have endeavoured to demonstrate that the causal agent is a protozoan organism; but even these are too large to pass through a porcelain filter, so that if the organism is a protozoan it must have an intermediary stage or a sporulating form which is infinitely small. The disease must, therefore, be placed with foot-and-mouth disease, &c., as one of those due to an ultra-microscopic organism.

When once the disease breaks out in a dairy, it is apt to spread to the whole herd, unless precautions be taken against its diffusion, by segregation of all affected animals and the employment of separate attendants, particular care being at



all times taken that the milker of an affected cow shall not touch a healthy one. The milk should be drawn off with the teat syphon, and, if the mammary gland becomes much inflamed, fomentations may be necessary, with other treatment recommended for "mammitis" (see *Principles and Practice of Veterinary Surgery*); but in all ordinary cases of the disease non-interference is to be strictly observed.

The protective vaccination of human beings by using cow-pox lymph has, now that its benefits have received almost universal acceptance, been placed upon sound commercial productive lines, and this is accomplished in specially fitted laboratories, with a proper staff, whose duty it is to provide pure vaccine lymph obtained from healthy calves. The old method of vaccinating from child to child was objectionable and dangerous, from the risk of infection by other diseases. It is almost impossible to obtain lymph from a calf that is not contaminated with various pathogenic and non-pathogenic organisms unless elaborate precautions are taken. It is therefore better that good lymph should be prepared and issued from central laboratories. The power of glycerine to kill ordinary staphylococci, and not to in any way reduce the activity of vaccine lymph, has been taken advantage of in this preparation, and the *modus operandi* of lymph-production is as follows: Healthy calves of from four to seven months old are selected. It is well to test them with tuberculin, and to keep them under observation for some weeks prior to using them. Such a calf is secured on a clean table in an operating-room; its abdomen is shaved and thoroughly washed with hot water and soap, and then douched with sterile distilled water; no antiseptics are used, but if the above operation be properly performed, the abdominal surface will be sterile. Calf-lymph is then inoculated by scarifying the skin in longitudinal lines all over the shaved surface, taking care not to draw blood, as this would have a bactericidal effect upon the virus. The virus is well rubbed into the scarification with a sterile horn spatula, the calf released, and a sterile cloth bound loosely round the vaccinated surface. Five days after the operation the virus will have "taken," and be ready for removal. It is scraped off with a blunt sterile spoon quickly and cleanly, again taking care not to

remove any blood. The vaccine so obtained is mixed with glycerine and water, equal parts of each, and one part of vaccine to four parts of the mixture. The emulsion so produced is then thoroughly pounded in a special grinding apparatus, and is then ready for use. It is as well to allow a month before use, as the glycerine will ensure the destruction of extraneous organisms in that time. It is understood that every stage in the operation is conducted with absolutely sterile materials. The vaccine for issue is put up in small capillary tubes, each containing some six to eight doses.

The calves recover from the effects in a few weeks, but should be destroyed, and a *post mortem* made to see if they are free from all infection. By this means, if any calf be found diseased, the lymph should be discarded.

There is now no doubt that small-pox and cow-pox are one and the same, and that the former is modified in its passage through the latter, so rendering it a useful vaccine. This fact is taken advantage of to obtain a more active calf-lymph for protective vaccination. A successful lymph may also be obtained in the same way from horse-pox.

## CHAPTER XXI.

### CONTAGIOUS DISEASES—*continued*.

#### VARIOLA OVINA (SHEEP-POX).

*Synonyms*.—*Claveau* or *Clavelée* (F.); *Schafpocken*, *Schafpochenseuche*, and *Schafblattern* (G.)

*Definition*.—A contagious and infectious eruptive disease, analogous to small-pox and cow-pox; runs a definite course, and occurs but once, as a rule, in the same animal. It is divided into two forms:—1st. A malignant, virulent, or confluent form; and 2d. A benign or discrete form. The malignant form never produces vesicles; the sheep lose their eyes; their wool falls off, the skin cracks in a zig-zag manner, and the nostrils become filled with a fœtid discharge. In the benign form, genuine vesicles appear, which, after the scabs fall, leave pits in the skin, on which the wool never grows again. According to Professor Simonds the disease is not communicable to the cow or to children. Saccho, however, states that *orination* is protective against small-pox.

#### HISTORY.

Sheep-pox is said by Mr. Fleming to have appeared as an epizootic in England about A.D. 1277, but that it was well known in Britain more than two hundred years previous to 1275, and was called the Rot. The term variola had, however, been made use of in 569 (*Animal Plagues*, 1875). But in more modern times sheep-pox was unknown in this country until 1847, when it broke out on a farm at Datchett, near Windsor, where it was introduced by fifty-six merino sheep, brought to this country in the ship "Trident," from Tønning, in Denmark.

Professor Simonds, in his treatise on Variola Ovina, says of

these sheep—"When disembarked they appeared to be in health, and were sold by the salesman to whom, with other sheep, they were consigned. We have not succeeded in tracing the subsequent distribution of each separate lot of this cargo, but we are assured that many of the animals continue well. It is, therefore, evident that in this particular instance the malady was imported from Denmark; but this unfortunately did not prove to be a solitary case of its introduction.

"Within a day or two of the arrival of the 'Trident,' the 'Mountaineer' and the 'Princess Royal' came into port, each vessel having on board a number of merino sheep brought from Hamburg. . . . Some of these sheep—507 brought by the 'Princess Royal'—were sold to butchers; others to farmers, as 'stock sheep.' The latter were bought in several quantities on 26th July by Mr. B. Weall, of Woodhall, Pinner; Mr. Goodchild of Kingsbury; Mr. Pittman of the same place; and Mr. Choke of Barking. These purchased by the two last-named gentlemen are said to have continued in health ever since. Mr. Goodchild's sheep, however, showed symptoms of the disease very shortly after arriving at his farm, their illness being attributed to his having had them dipped or washed, for its true nature was not suspected. That portion of the cargo of the 'Princess Royal' which was purchased by Mr. Weall consisted of 80 merinos; and on the same day he bought 166 other sheep of the merino breed that had arrived by the 'Mountaineer.' These two lots were placed together, and subsequently, being sent to Pinner, were equally divided between Mr. Weall and his brother. In each moiety the disease showed itself, being first observed among the 80 sheep about ten days after their purchase, and from them it rapidly extended to others. Out of those belonging to Mr. B. Weall, 20 died in the acute stage of the malady, 27 more were sacrificed, and the residue was disposed of at a low price."

"We examined Mr. B. Weall's flock on the 7th of September, and found *two of the sheep* in the *earliest* stage of the malady; but most of them were recovering."—(SIMONDS on *Variola Ovina*.)

The outbreak of 1847 committed great ravages in Middlesex, Surrey, Norfolk, Cambridgeshire, Suffolk, and Hampshire, and continued during 1847, 1848, 1849, and 1850; the losses from

it were also severe in 1852, and more so in 1853, at which time the practice of slaughtering sheep in or near London put an end to the disease until 1862, when it broke out at Allington, in Wiltshire. This outbreak was supposed to be of spontaneous origin, as it could not be distinctly traced to foreign sheep. Mr. Gamgee visited the district at the time, and made many observations, from which he came to the conclusion that "the district turns out to be one not unfrequently visited by contagious disorders. . . . The district is traversed in all directions by driftways, so that drovers can pasture their sheep on the downs for days, and go from Bristol to London with the payment of a single toll. There are some notorious dealers who have no farm or down on which to keep their flocks. They pick up odd animals at a low price here and there, and drive over the downs, where they sleep, and move gently backwards and forwards on the pretext of travelling, but in reality getting food for their flocks. Many instances have occurred of the spread of contagious disorders, such as scab, the foot-and-mouth disease, foot-rot, &c., from these infected flocks passing over the downs along the Wans Dyke." The disease is not readily transmissible to other animals, but it appears that goats, pigs, hares, rabbits, and dogs are amenable to it.

These observations of Mr. Gamgee, along with the fact that sheep-pox is one of those diseases which never occur spontaneously in this country, are to my mind quite sufficient to account for the Wiltshire outbreak.

The early history of sheep-pox on the Continent of Europe, in some parts of which it is very prevalent, is very confused; it is certain, however, that it was introduced into Western Europe by diseased animals brought across the Russian frontier into Poland, Hungary, Prussia, Pomerania, &c. Rammazini states that the malignant affection amongst cattle and sheep which prevailed in 1514, 1599, and 1691 was small-pox. From his description it is very probable that the disease of 1691, which chiefly attacked sheep, was in reality variola ovina, inasmuch as it was characterised by pustules similar in form, colour, and in the manner in which they went off. When they had died off after the suppuration, they left a black scar, like that which remains after the small-pox.

Professor Simonds instituted various experiments which prove

that variola ovina is inoculable, contagious, and infectious, and that its incubative stage varies from seven to thirteen days when communicated by inoculation, and from nine to twelve days in *natural* cases.

Like the poison of cattle plague, that of sheep-pox is both volatile and fixed, existing in the blood, the tissues, secretions, and emanations, and may be carried from place to place in the clothes of attendants, in fodder, wool, skins, and particularly by dogs. Mr. Chas. Percivall informed Mr. Gamgee that he had seen a dog (Mr. Stephen Neate's) suffering from symptoms of disease identical with those of variola ovina, and there could be no doubt but that the dog contracted the disease from affected sheep (*Domestic Animals*); and it is pretty well demonstrated that a healthy flock is not secure from the disease at the distance of a quarter of a mile from affected sheep. Upon this head Continental writers are very clear, and they point out the danger of driving a healthy flock on the same road which shortly before has been travelled over by a diseased one. Chauveau has shown that the virus is a hundred times more infective, that the matter contains in an equal quantity thirty times the number of corpuscles, and is more active than that of vaccina. He has also shown that if vaccine matter be diluted with fifty times its weight of water, it propagates itself when inoculated with great uncertainty, while ovine matter may be diluted 1500 times before its virulence is appreciably affected.

The virus retains its vitality, when protected from the air, for a very considerable period, but is destroyed by the influence of air, heavy dews or rain, sunlight, a temperature of 122° F. frost, and disinfectants, such as chlorine, permanganate of potash, and ozone, as well as by putrefaction, and if the wounds be suppurating, the matter from them will not infect.

#### PATHOLOGY AND SYMPTOMS.<sup>1</sup>

As already stated, the period of incubation varies from about the seventh to the thirteenth day, during which time there are no symptoms observable to a non-professional person. Like all specific fevers, however, there is an increase in the temperature of the animal body for some time prior to the outward

<sup>1</sup> The remarks made concerning the etiology of small-pox apply equally to sheep-pox. It is one of those contagious diseases due to an organism of the ultra-visible variety. Sheep-pox would appear to be as malignant a form amongst ovines as small-pox is to man

manifestation of the disease. In thirty-six to forty-eight hours after the first manifestation of the disease, the temperature rises to  $105^{\circ}$ , or even as high as  $107^{\circ}$  or  $108^{\circ}$ , but in the benign form it diminishes at the commencement of the eruption. In the malignant it is slower and later in diminishing, more irregular, and increases when suppuration occurs, becoming normal at the period of desiccation. A careful observance of the temperature is of great importance, in order that affected sheep may be removed from the flock before any of the ordinary signs of the malady can be detected, and before the *disease becomes infectious*. By carefully watching and examining their flocks, some farmers have been able to arrest the spread of the malady, and the course they have adopted has been that of segregating the diseased ones when the slightest rash made its appearance upon them. Mr. Charles Fielder of Sparsholt, near Winchester, says—"I employed two men to turn every sheep I had on my farm, and minutely inspect every one of them; and if they saw the slightest appearance of any rash, or a single pustule showed itself between the inside of the shoulder and the breast, where the skin of the sheep generally looks white and clean, and where it was sure to show itself first, *I had it immediately taken away, putting the whole of the diseased together in one large field in the middle of my farm, at a distance from any road, as a protection to my neighbours*. I followed the same course every morning, by having all my sheep turned and closely inspected, not looking to the trouble and expense, as I felt in my own mind that it was the only remedy I had to stop the *infection*; as I calculated, if I could only find out those sheep which had taken the disease and were breeding it, *before it became infectious to others*, I should be able to arrest its progress. Fortunately I was right in my calculations, for they daily decreased in numbers, although they still kept faltering for a fortnight or three weeks from the time I began turning them (particularly in one flock where the Spanish sheep had been), before it, as I hoped, ceased altogether, as I have not found a diseased one for some time past."—(From the *Farmers' Magazine*, vol. xvi., page 524, quoted by Professor SIMONDS.) If separating the diseased from the healthy sheep after the first symptoms of the malady are discernible by an ordinary observer has so far been sufficient to arrest its spread, it can easily be understood that removal prior to the appearance of the rash will yet prove more satisfactory.

*Symptoms.*—The first appreciable signs of the disease, after the termination of the incubative stage, are recognisable “by the existence of papulæ or nodules deeply imbedded in the dermis, having a florid red aspect. These show a preference for some parts of the integument, and are usually to be first detected on the inner side of the arms and thighs, and on the cheeks and lips, where the animal has a hairy and not a woolly covering. Other portions of the body are often simultaneously attacked, as the prepuce, labia, anus, and under surface of the tail—parts which are perfectly nude.”—(SIMONDS.)

In some cases there will be a diffused redness of the skin, in patches of various sizes, prior to the formation of the papulæ. Professor Simonds says that in inoculated cases this is so well marked that it constitutes a disease of the skin, analogous to *roseola variolosa* of the human subject, and establishes another similarity between these maladies.

During the papular stage the skin has a flea-bitten appearance, the eruption being at first in the form of small red points; these gradually enlarge, often uniting one with another. Professor Simonds considers that three days may be regarded as the period of papulation, and that it is generally longer in confluent than in the distinct form of the disease. The disappearance of the papulated eruption, the disease afterwards lying dormant in the system for three weeks and then breaking out, as described by Hutrel d'Arboval, was not witnessed by Professor Simonds.

Concomitant with the appearance of the rash, febrile symptoms are manifested, the animal becomes dull, with blood-shot eyes, is thirsty in all cases where the eruption is extensive, and the papulæ confluent. Where the eruption is distinct the febrile symptoms are much milder. Continental writers state that the outward signs of fever precede the eruption: the experience of Professor Simonds and others in this country does not confirm this, for in all cases seen by Professor Simonds the eruption preceded the constitutional disorder.

“The red papulæ gradually enlarge in size, then become elevated and transparent in their centres. The papula is now a vesicle containing a liquid at first transparent and then turbid. Many of the vesicles are very small, and if punctured yield no fluid. The duration of the vesicular stage differs to some degree, and it has been observed that many of the papulæ disappear without vesicles being produced. The ovine vesicle is flat on its



surface; and in this particular it forms a contrast both with the vaccine vesicle of the cow and the variolous of man. The serosity of the vesicles, first clear, becomes milky, turbid, less serous, and straw coloured, and ultimately by drying hardens into a crust, and is cast off with the epidermis.”—(SIMONDS.)

Both Professor Simonds and Mr. Ceely are of opinion that the term *pustule* should not be applied to the eruption, except, as Mr. Simonds says, in the latter stages of extreme and protracted cases, where pus is in reality formed, and is succeeded by the ulcerative process.

“The period of desquamation or desiccation depends on the extent of the original eruption, and also on its being distinct or confluent. It is likewise governed by the amount of inflammatory action which existed in the dermis, and is always protracted when suppuration has taken place.”—(SIMONDS.)

The vesicles, scab, and crusts are liable to be injured in various ways, but more particularly by the sheep scratching themselves, and this interferes materially with the process of healing, and causes considerable irregularity in the duration of the malady. “In natural cases, even when mild,” says Professor Simonds, “and when no cause retards their completion, a month, reckoning from the period at which the animal was first exposed to the contagion, usually passes before it is restored to health.

“The following summary of the gradations of the malady may be accepted as sufficiently accurate for practical purposes:—The first ten or eleven days are those of *incubation*; the twelfth and thirteenth of *invasion*; the fourteenth, fifteenth, and sixteenth of *papulation*; the seventeenth, eighteenth, and nineteenth of *vesication*; the twentieth, twenty-first, and twenty-second of *suppuration*; and the twenty-third to twenty-eighth of *desiccation* and *separation* of the crusts.”—(SIMONDS on *Variola Ovina*.)

The same authority says that the constitutional symptoms, especially those which indicate the greatest danger, “are dejection, the head being held low; the infected animals separate from their fellows; mostly lie down in a corner of the field; the ears are pendent; the breathing quick and short; the eyelids are swollen, and tears trickle down the face; the conjunctiva varies in shade from a bright scarlet to a modenared; a mucous discharge flows from the nostrils, and increases in viscosity as the disease advances, often becoming sanguineous in the latter stages; yellowish spots may likewise be seen scattered

here and there on the pituitary membrane; the pulse early gives evidence of febrile excitement; at first it is quickened and somewhat increased in force, numbering about ninety-five beats in a minute; later on it becomes tremulous and indistinct, even over the region of the heart; rumination is suspended, and all food refused; but the patients show a great disposition to take water, and also to lick earth, which, mingling with the mucus of the nose, assists in clogging the nasal openings, and renders the respiration more difficult. The alvine evacuations are but little changed in the majority of cases; in some, however, diarrhoea succeeds to a torpid condition of the bowels, and hastens the fatal termination; the temperature of the body is unequal, the feet and ears being generally cold, while the skin elsewhere is hot.

“These symptoms, more or less modified, are present from the commencement of the eruption, and seldom abate until the papular stage gives place to the vesicular, when the animals usually experience a relief. In all natural cases, the constitutional disturbance is great, and makes the probable termination of the attack doubtful, which is also rendered more uncertain when the confluent form of variola prevails; for the type of the disease, as elsewhere stated, governs to a considerable extent the amount of the fatality.

“Not only is the prognosis unfavourable in confluent variola, but it is equally so if the fever continues unabated, which is certain to be associated with an augmentation of the patient's sufferings. The breathing now becomes rapid and painful; the inspirations frequently, but more often the expirations, are accompanied with a moan; frothy saliva is discharged from the mouth; the exhalations are foetid; the wool separates from the skin on the application of the slightest force; ulcerations of the conjunctival membrane occasionally result; and sometimes the internal structures of the eye give evidence of disorganisation; the animal shrinks from the touch; the pulse gets more and more indistinct; great prostration of all the vital powers succeeds, and death closes the painful scene.”

The percentage of mortality arising from variola ovina varies considerably; very often half the sheep which are attacked succumb to the disease, and it is not unusual for nearly the whole flock to be swept away. Mr. Gamgee states that in one outbreak “the losses on 3811 sheep were 221 before inoculation

had been practised, and the loss since has amounted to 537, or a total of 758 on 3811, viz., very nearly 20 (19·89)<sup>1</sup> per cent., whereas in 1310 not inoculated the loss amounted to 21 sheep, or 1·6 per cent.”

It is evident that Mr. Gamgee intends the reader to understand that the small mortality in the 1310 was due to the precaution of separating the diseased from the healthy animals during the earlier stage of the malady.

#### POST MORTEM APPEARANCES.

The *post mortem* appearances of small-pox are as follows:—Body swollen, and exhaling a foetid odour; the eyes and nose closed with dry discharges; the mucous membranes of the mouth, nose, pharynx, cesophagus, larynx, bronchi, and rumen are covered with light copper-coloured *vari* or nodules, and occasionally small ulcers are seen on the epiglottis and other parts of the larynx. The lymphatic glands in various parts of the body are enlarged, and marked with red spots. The subcutaneous tissues are engorged with blood, and covered with solid red nodules, whilst purulent formations exist beneath the confluent papulæ. The areolar tissue of the face and extremities is often filled with effusion, and the skin itself is in some parts red, in others of a blackish hue, and the wool, if not already detached during life by the animal scratching itself, easily comes off.

#### PREVENTION AND TREATMENT.

The prevention of the disease in this country depends upon the exclusion of animals from infected countries. The incubative stage being of such a duration, affected animals may easily pass the examination of the most careful inspector, no signs of illness being manifested during this stage.

If, however, variola should appear in a flock, the best way of dealing with it is that recommended by Mr. Fielder (see page 214), namely, the separation of all diseased sheep during the earliest stage of the disease; and pastures, roads, and other places frequented by the diseased animals should be avoided until heavy rains have destroyed the contagium.

<sup>1</sup> In Mr. Gamgee's book it says very nearly 10 (19·89) per cent.; the figure 10 is evidently a mistake.

Inoculation or ovination is recommended by very high authorities. In this country, however, the experiment has not proved at all satisfactory. French veterinarians speak highly of ovination: thus, at the Alfort Veterinary School the mortality was only at the rate of 1 in 400 after ovination. Professor Simonds says our experiments are too limited to suggest correct conclusions, and they have shown a result so different, that, were we to found an opinion on the merits of ovination on them alone, it would not be in favour of the practice. The deaths have been at the rate of 20 per cent. Professor Gamgee condemns ovination entirely. Undoubtedly inoculation serves to increase the centres from which the contagium may extend in every direction, whereas segregation or slaughter at once diminishes the cause of the propagation of the disease, as well as the centres from whence it might spread.

Variola ovina, being a disease that runs a definite course, is not beneficially influenced by the action of medicines; on the contrary, the forcible introduction of drugs, or even of gruels, and other so-called nourishments, into the animal's stomach when it is incapable of digesting them, is calculated to do much harm. If the diarrhoea be excessive, chalk is to be mixed with the water which the sheep may drink; or if there be much fever, but no diarrhoea, chlorate or nitrate of potash may be ordered in the same vehicle. It is essential, however, that the sheep be kept clean, have their nostrils sponged or syringed to prevent suffocation, have clean beds to lie upon, be well sheltered and kept warm, have plenty of fresh air, and, if they eat at all, be supplied with easily digested food.

On the Continent variola occurs among goats, pigs, dogs, and fowls.

Mr. Gamgee describes a form of *chicken-pox* in cattle, occurring on the Continent under the term *varicella boum*.—(See GAMGEE'S *Our Domestic Animals in Health and Disease*, p. 256.)

#### VARIOLA EQUINA, OR HORSE-POX

(Also called grease, constitutional grease, Soy's disease, and sore heels), is described by Dr. Fleming in his *Sanitary Science and Police* as an eruptive vesico-pustular malady, generally diffused in the different parts of the world inhabited by the horse. With the exception of the case mentioned at p. 193, I have never witnessed anything approaching to variola in the horse.

It appears, however, that the eruption, preceded by a very slight, almost imperceptible, degree of fever, may appear on the skin at different parts, or over the whole body, on the nasal and buccal mucous membranes, and occasionally on the conjunctivæ.

The nasal eruption not only involves the Schneiderian membrane, but the nostrils and lips, and is apt to become confluent on the lips and the inferior parts of the limbs.

The contagium of the variola is transmissible from horse to horse, to the cow and to mankind, by contact and inoculation but not by infection.

*Variola equina* appears to be a very mild and benignant disease; and as Dr. Fleming's work contains a full description, the reader is referred to it, as well as for descriptions of the goat-pox (*variola caprina*), porcine pox (*variola suilla*), *variola canina*, and the variola of fowls. Professor M'Eachran, of the Montreal Veterinary College, reports in the *Veterinary Journal*, August, 1877, an outbreak of equine variola in Montreal in February and March of that year.

*Variola equina* has been confounded with grease, but there is not sufficient evidence to show that the two conditions have any relationship. Grease may be excited by variola, but the former is a comparatively common affection, and the latter is very rare, at least in England. Some prominence has recently been given to variola in horses in India by Pease and Baldrey, on account of its having been compared with dourine. The variolous eruption is mild, and often affects the genital organs of entire horses and breeding mares, and this eruption in its pustular stage has given rise to the fear of a dourine infection. Its evanescent character and mildness, and the absence of any trypanosomes, however, render it not difficult in differential diagnosis.

Horse-pox in a somewhat acute form occasionally occurs in an epizootic form amongst horses, and attacks the nostrils and lips often without affecting either the coronet or the genitals. In the pustular stage, ulceration may supervene and spread into the mucous membrane of the nose. In this case it has happened that glanders has been suspected; but again, the rapidity with which the lesions heal, and the absence of any other glanders lesions, will render diagnosis easy, even without the use of mallein.

## CHAPTER XXII.

### CONTAGIOUS DISEASES—*continued.*

#### RABIES.

*Definition.*—A disease originating in the canine, and, less frequently, in the feline race. During the progress of the malady a specific virus is developed in the saliva of the affected animal, which, being implanted through a wound, bruise, or thin epidermis without wound or abrasion, conveys the disease to other animals and to man. After an indefinite period of latency, it causes pain and stiffness in the bitten part, excitement, feverishness, inability to swallow liquids, a tendency to bite, great prostration, and death. The poison is only recognised by its morbid effects in the animal economy.

#### HISTORY.

Rabies (*Rabio*, to rave), or, as it is sometimes erroneously called, so far as the lower animals are concerned, hydrophobia, is a disease which has been known from very early times.

Dr. George Fleming, in his work on Rabies and Hydrophobia, says—"Its great antiquity is undoubted. Plutarch asserts that, according to Athenodorus, it was first observed in mankind in the days of the Asclepiadæ, the descendants of the god of medicine, Æsculapius, by his sons Podalirius and Machaon, who spread through Greece and Asia Minor, as an order of priests, prophets, and physicians, preserving the results of the medical experience acquired in the temples as a hereditary sect. They were the earliest physicians known to us, and it is not unlikely

that they may have been the first to observe the madness of dogs transmitted to man." Dr. Bardsley, in his Memoir on Hydrophobia (*Cyclopædia of Practical Medicine*, vol. ii.) says—"We have already said that the disease was well known to Homer, and applied by him, with his usual critical exactness of similitude, to the indiscriminate havoc with which Hector sweeps through the battlefield of his enemies."

Hippocrates alludes to hydrophobia in the faintest manner; but Democritus, who was contemporary with Hippocrates, according to Cælius Aurelianus, wrote upon the nature and treatment of the disease. Aristotle, in his *De Historia Animalium* says that the disease is communicable to all animals but man. Many other ancient and modern writers have described the fearful malady: amongst the latter may be mentioned Van Swieten, Sauvages, Cullen, Mead, Bardsley, Youatt, &c.

In 1271 wolves became affected with rabies in Franconia, and, contrary to their usual habits, they spared the herds and flocks and attacked human beings: upwards of thirty men fell victims to these attacks. In 1590 canine madness prevailed in Spain—(BLAINE.) In 1590 epizootic rabies prevailed amongst the wolves in the province of Monthelliard. The further history of rabies may be obtained from Dr. Fleming's excellent treatise, to which the reader is referred.

Like other epizootic and contagious diseases, its prevalence is liable to many fluctuations, at times prevailing to a great extent, whilst for long periods the disease is almost unheard of. During the earlier portion of the present century rabies was very prevalent both in this country and on the continent of Europe, not only in dogs, but in foxes as well. Since then there has been an abatement in the frequency of the disease; but even up to the present, outbreaks are not very rare. In 1869 I witnessed the disease in a pack of hounds in the north: the nature of the disease was detected after one man had been bitten, and he fortunately resisted the action of the poison, although he took no precaution against its effects. The disease has again been prevalent in 1876-7-8 in the southern and lowland counties of England.

By the stringent rules enforced by the Board of Agriculture, rabies has now been stamped out in England, Scotland, and

Wales. Occasional cases are found in Ireland, but it is there rapidly on the decrease. Rabies is a disease which owes its prevalence to the fact that it is spread only by actual inoculation, and this can only be accomplished by animals addicted to biting. In Britain the dog is essential, and it therefore is a comparatively easy matter to suppress the disease. If there were no dogs, wolves, jackals, or other similar carnivora, it is very probable that there would be no rabies. Britain, on account of its insular position, can be easily protected against the disease. The Board of Agriculture prevents the importation of it by a strict quarantine of six months on all imported dogs; and stamped out the disease in Britain by stringent muzzling regulations applying to dogs, and immediate slaughter or isolation of all suspects or strays. These conditions are not easily accomplished in foreign countries, where carnivora, such as wolves, jackals, &c., exist in a wild state, and spread the disease to one another, and also to other animals and man.

#### CAUSES.

Opinions have been divided as to the spontaneity or otherwise of the origin of rabies. Some maintained that even in the dog and allied quadrupeds the malady never originated except by inoculation; whilst others held that it originated spontaneously in the dog, even in this country.

After discussing the opinions of Blaine, Youatt, and Maynell, who affirm it owes its origin to a wound from a rabid creature, Fleming says: "There are few nowadays who are not convinced that it will occasionally appear in a spontaneous manner, and without any certain assignable cause. No doubt the transmission of the disease by inoculation furnishes by far the largest number of cases, and many of these, from the obscure manner in which the inoculation has been effected, appear to be due to other causes than that of a traumatic character; but, notwithstanding, the disease must have a commencement." However, he changed his opinion, and affirmed that it originates from contagion only. In this view of the origin of the malady, facts and circumstances compel me to agree with Dr. Fleming; at the same time I am fully aware that the opinions of many observers are opposed to it. Professor Dick was of opinion



that rabies always originated spontaneously—from atmospheric causes—a species of catarrh, which, by extension from the nasal mucuous membrane, through the cribriform plates of the ethmoid bone, involved the meninges of the brain, and produced the phrenzy, delirium, and the other train of nervous symptoms which characterise the disease. He also held the opinion that it was never caused by inoculation from the bite of a mad dog; in fact that it was a non-contagious disease.—(*Veterinary Papers* by Professor DICK.) This opinion of Professor Dick is still believed by a few, but fortunately very few, and the sooner it is dispelled the better, for nothing is more calculated to do mischief than the promulgation of what has been proved to be so erroneous. Rabies, once generated in the dog or cat, is transmissible by inoculation to every warm-blooded creature.

The idea that hot weather is productive of rabies is also now dispelled. The following report, arranged by Professor St. Cyr, of the Lyons Veterinary School, is very instructive, showing that rabies is even more prevalent in the temperate months than in those of extreme heat and cold:—

	Rabid.	Suspected.	Became Rabid in Infirmary.	Total Admitted.	Total Rabid.
January, .	12	2	0	14	12
February, .	14	4	1	18	15
March, .	6	11	0	17	6
April, .	14	20	1	34	15
May, .	12	14	1	26	13
June, .	6	14	1	20	7
July, .	2	8	2	10	4
August, .	7	3	2	10	9
September, .	1	3	0	4	1
October, .	3	1	0	4	3
November, .	0	0	0	0	0
December, .	1	1	1	2	2
	78	81	9	159	87

Sometimes the annual statistics show a predominance of cases in the summer months. But according to M. Bouley's observations there is no great difference in the seasons with regard to the disease. An analysis by M. Bouley of reports

for the six years 1863-1868 gives—for the spring months, March, April, and May, eighty-nine cases; for the summer months, June, July, and August, seventy-four cases; for autumn, September, October, and November, sixty-four cases; and for the winter months, December, January, and February, seventy-five cases.

It was at one time maintained that rabies never became developed in the bitch, and it is now advocated that it is far less frequent in females than males. Professor Coleman stated in evidence in 1830 that on the occasion of rabies entering kennels, the mad dogs bit dogs but spared the bitches. This opinion of Coleman is still believed in, more particularly by medical writers. But there can be no doubt that bitches are as susceptible to the contagion by inoculation as dogs. This is well exemplified in what occurred a few years ago to a bitch pack of foxhounds belonging to Mr. Standish, South Shoreham, Southampton. The pack was nearly exterminated by rabies, through the disease having been introduced to the kennels by a hound which had strayed away for several days, and on its return was discovered to have been bitten. — *Veterinarian*, vol. xlv.

I think the reason for the supposition that fewer cases of rabies are seen in bitches than in dogs, is to be found in what seems to have escaped medical observers, namely, that there are fewer of them in the world. Indeed, the same explanation can be given on this point as on what appeared so strange to some during the prevalence of the cattle plague, that fewer bulls than cows were attacked by that malady.

There no longer exists the slightest doubt that rabies can originate in any other way than from a previous case. Spontaneous origin is impossible, and in some of the cases in which no previous bite or method of infection could be traced, it is from lack of an exact history that the idea of spontaneity has arisen.

The *materies morbi* of the disease are found in all the body fluids, and especially in the saliva, which becomes infective as soon as any symptoms appear. What the actual infection is we do not know. It is, however, an extremely small organism, or one capable of extremely small phases in the course of its development, as virulent saliva when

filtered through a fine porcelain filter is still infective. Many observers have advanced arguments as to the actual contagion, but none are so far satisfactory, and it is only safe to say that the disease is one due to an ultra-microscopic organism.

The theory of its protozoan origin has been advanced, and it has been found in the spinal cords of affected animals, and in the cerebellum, fons of Ammon, and origin of the seventh nerve bodies, which are called Negri bodies. These are small ovoid cellular bodies, varying in size from 10 to 25  $\mu$  long and 1 to 5  $\mu$  broad. They are dark, covered with dots of a darker shade; they stain with some difficulty, but are easily seen in very thin sections of the medulla with a high power. They are too large to pass through a filter, and, therefore, cannot be the form in which the organism causes the disease; and if they are the cause, they must have some other period in their life-cycle in which they are smaller. These bodies are also found in the medulla of very old dogs, so that it seems probable that constant existence in connection with rabies is accidental, and that they are the products of some nerve disintegration. No other specific cause has been ascribed, and until the Negri bodies have received further attention it is not possible to accept them as such, although their constant presence has rendered diagnosis practically certain if they are found. This is a very considerable advantage, as formerly until a rabbit died it was impossible to give a positive diagnosis. By section and examination of the cord, this can now be given in a few hours.

#### PATHOLOGY AND SYMPTOMS.

The following circumstances in the pathology of rabies are worthy of notice, namely—*First*, That the period of latency after inoculation is very indefinite, and that it varies in different animals; *Second*, That inoculation does not always produce the disease, one-fourth of the inoculated animals generally escaping; and *Third*, That, notwithstanding the strongest evidence as to the microbic nature of the disease, the morphological character of the microbe is as yet undeter-

mined. Cocci observed in the spinal cord of rabid dogs have been described by Babes (*Les Bacteries*, 1886), cultures from which were said by Babes to communicate the disease. Babes also describes a short bacillus. And again it is said that very fine bacteria have been isolated from the brain, cultures from which gave the disease. Researches are being still carried out which will in time doubtless determine the nature of the microbe. Virulence is said to be destroyed in thirty hours at a temperature  $-20^{\circ}$ , and in moist heat at  $100^{\circ}\text{C}$ . in half an hour, and desiccation in the air diminishes and even destroys the virulence in a few days. This fact has enabled Pasteur to modify his method of preparing his vaccine, which at first was transmitted from the dog to the monkey, in which animal the virulence became enfeebled after each transmission, until it became too attenuated to convey the disease, and was used as a preventive vaccine. Now, however, Pasteur inoculates a rabbit, in which the period of incubation is always short, through a trepan in the skull, having discovered that after successive inoculations from one rabbit to another virulence becomes intensified, and the period of incubation shortened, until it is reduced to six days. The virus then has attained its highest degree of virulence, and is called "virus fixe." The spinal cord of a rabbit, having had this intensified rabies, having been suspended in a jar at a temperature of  $73^{\circ}\text{F}$ . in an atmosphere kept dry by the presence of potash, gradually loses its virulence. The opinion that rabies could not be transmitted after an animal had been dead twenty-four hours, or after *rigor mortis* had been completely established, has been found to be erroneous; for it is now placed beyond doubt that virulence may be retained for a considerable time, provided putrefaction be prevented. The activity of the virus is also destroyed by various disinfectants, as such 1-1000 corrosive sublimate solution, 5 per cent. solution permanganate of potash, 50 per cent. alcohol; but 15 per cent. alcohol is said to preserve it for several days, and large doses are rendered innocuous by being rendered acid, alkaline, or putrefactive. The virus of rabies is contained in its most concentrated form in the saliva of the rabid animal; but it by no means follows that other parts of the diseased animal are free from its presence; indeed, direct experiments have determined that the virus is present in

the blood, flesh, and other parts of the rabid animal. The virus of the rabid skunk, wolf, and hyæna appears to be more virulent and prompt than that of the dog.

It was at one time believed that the virus of rabies was innocuous when the bite was inflicted by any of the herbivora. Observation on accidental cases had led to this belief, and direct experiments seem to confirm its correctness. Girard, Vatel, Huzard, Dupuy, Lafosse, and others failed to transmit the disease from the herbivora to the dog. Professor Coleman, Sir Astley Cooper, and others in this country believed that the disease was not communicated except by animals which "naturally employed their teeth as weapons of offence." Dr. Fleming mentions several instances where rabies was communicated by inoculation with virus obtained from cows, sheep, and even domestic fowls, and argues "that the facility with which the disease can be transmitted by different species depends, besides the activity or degree of virulency of the infecting principle, upon the organisation, habits, or rather nature of the disease. Flesh-eating or carnivorous animals, as is well known, generally attack other creatures with their teeth, which are well adapted for wounding and tearing; consequently they are the most successful in inoculating with the poison."

The virus, having been introduced into the system, generally lies dormant for a very indefinite period of time, the shortest period in the dog being about seven days, and the longest 155 days, but in rare exceptions even longer than this; in man, from three days to nine months, and even ten years in rare instances (such cases, if inquired into, will show a subsequent inoculation); in the horse, from fifteen days to three months, and even fifteen months; in the sheep, from fourteen days up to three months; and in the pig, from a few days—eight or nine—to as many weeks, or even months. The wound inflicted by the rabid animal generally heals rapidly. In 1862 M. Renault published the results of some experiments which had been conducted with the view of determining the period of incubation in the dog. Out of 131 dogs bitten by mad dogs, or inoculated with their saliva, 63 remained well at the end of four months; the disease being developed in the other 68 after intervals varying from 5 to 120 days. Thus—

In 25 dogs				the disease set in between the 5th and 30th day.
In 31	„	„	„	30th and 60th day.
In 7	„	„	„	60th and 90th day.
In 5	„	„	„	90th and 120th day.

*Symptoms in the Dog.*—There are no premonitory signs in the lower animals, at least none that can be detected—such as pain in the seat of the bite, melancholy and irritability, febrile disturbance, and stiffness about the neck and head. The dog, when the period of latency is passed, becomes restless, dull, watchful, withdraws from its companions, choosing solitude, shunning the light; hiding in corners, or below chairs or other furniture; being fidgety, lying down, then jumping up again in an excited, unnatural manner; it has a tendency to rove about, and if possible to escape from its room or kennel, and wander about the country. At first, the dog's power of recognising people about it does not seem lost; indeed there are moments when the usual faculties of the dog, its affection and liveliness, seem greater than usual. Along with these symptoms there is a desire to lick anything cold, to rest the nose on a cold object, and to pick up stones, bits of wood, straw, &c. At this stage the tendency to bite is not observed, but as the disease advances the movements of the animal become unsteady; the eyes follow objects in a peculiar staring manner; sometimes the dog will stare at some imaginary object, then rush forward, and bite at anything that may chance to be in his way, or even at the air, as if he were catching flies; cushions, straw, and other objects by which dogs are ordinarily surrounded, are tossed about; the animal scratches the ground, snuffles as if on the scent; the appetite is lost, but the thirst is considerable, and the *act of drinking* is performed *without much difficulty* during the early stages of the disease. At a later stage, however, swallowing—deglutition—is performed with difficulty, or is not performed at all, owing to the spasmodic constriction of the throat; but even then the animal has no dread of water, does not shrink at the sight of fluids, but, on the contrary, will plunge its muzzle deeply into it, and endeavour to drink with great avidity. The coat is staring, the skin tight on the ribs, and the belly tucked up. The dog occasionally vomits, the ejected matter being tinged with blood; there is a brownish coating on the tongue; the nose and

mouth are foul and offensive, and there is generally a flow of saliva from the mouth. A symptom is mentioned in connection with the disease in the human being, which is of doubtful presence in the lower animals, namely, the formation during the early period of the disease of *lysses* or eruption on each side of the tongue. If any eruption does occur, its presence is seldom detected.

The mad dog, or indeed a rabid animal of any kind, brought into the presence of one of the canine species, exhibits great excitement, exasperation, and fury, with a desire to attack and destroy what seems the object of its hatred. "The horse assaults it with its teeth and hoofs; the bull, cow, and ram with their horns; even the timid sheep, when rabid, becomes the assailant."—(BOULEY.) The females of the canine species do not always lose their maternal affection; on the contrary, the young are attended to with great affection. In the course of a day or two after the first manifestation of the above symptoms, the characteristic signs become more marked; the desire to bite is greatly exaggerated; the pupils are dilated; the conjunctivæ red and injected; the eyes alternately widely opened with fury, and then closed in a dull but fierce manner. The forehead becomes wrinkled, and the looks of the animal are terrifying and repulsive; the presence of a living object excites the rage of the sufferer, causing it to spring at and endeavour to bite it. Any shining object will bring on a paroxysm of rage and excitement, and water, if the light shines upon it, will do the same; but if placed in a dark place, or where light does not shine, the dog will endeavour to drink with avidity. Intermitting with the excitement are periods of great prostration, the exhausted animal lying down in the quietest spot it can find, insensible to all surrounding objects. All at once, however, it springs up, and becomes greatly agitated; the excitement, rage, and agitation being always much greater when the animal is surrounded by noises and objects; when away from these things, in a quiet place, the fits of rage are not so great, indeed sometimes scarcely observed.

The bark of the rabid dog is unnatural; it is husky, spasmodic, and more of the nature of a howl. In some cases, the nervous symptoms are those characterising paralysis of the jaws, with inability to close the mouth; the lower jaw is dropped, the

cavity of the mouth and its contents exposed, the tongue dry, and the buccal membrane of a brownish hue. This is the "*dumb madness*," thus called because the animal is unable to bark or howl. Rabid dogs have a strange tendency to eat filth; they have been known to eat portions of dead dogs, and to swallow hair, coals, earth, excreta of all kinds, and these remaining in the stomach present a characteristic *post mortem* appearance. A mad dog, when loose, will travel an immense distance in a short time, generally rushing at everything that comes in its way, but preferring to attack other creatures than man, and finally endeavouring to return home, as if prompted by some instinct. The pulse is said not to be accelerated, nor are the respiratory movements much increased, except during the paroxysms; the bowels are constipated, and the urine is high-coloured; the gait is unsteady, the tail droops, the head is depressed, the nose protruded; the fits of rage become shorter, those of depression longer; scent, sight, and hearing are much impaired; insensibility to pain, which is generally present from the earliest stages, is more and more manifest; and finally paralysis, particularly of the hind limbs, supervenes, and if not destroyed, the dog dies, an emaciated, repulsive object, the desire to bite remaining to the last. In the dumb form, the ability to bite is lost, as well as the power to lap fluids. The exposed and protruded tongue is covered with a stringy, tenacious saliva; the desire to drink is even more intense than in the furious form, and attempts to lap and swallow become painful to witness. Squinting of one or both eyes has been observed; there is also abdominal pains in some instances, and an inflamed condition of the nasal cavities, larynx, and bronchial tubes. There is no tendency to harm and bite surrounding objects, and the dog dies from coma, exhaustion, and suffocation. Both forms of the disease terminate in death in from four to eight days. It is, however, reported that some chronic cases of rabies exist for an indefinite period.

With regard to the dread of water—hydrophobia—which has been looked upon by some as the diagnostic symptom of the disease, I may repeat that it is absent in the dog. The desire for water is always excessive; there is inability to swallow fluids, owing to the paralysed state of the pharyngeal muscles, hence a dog will plunge his nose deeply into water, lap it with avidity, but owing to the thirst being still unallayed, he will



become greatly agitated, and is thus often thrown into a state of fury or of involuntary spasms.

*Causes and Symptoms in the Horse.*—Rabies in the horse occurs from the bite or contact of the saliva of a dog or cat, generally from that of the dog. The disease is manifested in a variety of ways. In some cases it commences by great apparent distress, with sudden perspirations over the body; unruliness, the horse stamping and pawing violently, finally becoming frantic, and destroying everything within its reach. In other cases, along with restlessness, there is manifested a desire to bite the seat of injury. In one case that came under my notice, where the horse had been bitten on the breast, three months before any symptoms of rabies were visible, the animal during the paroxysms continually bit the cicatrix, until at last the whole sternal region was denuded of its skin; there was also intolerance to light, great nervousness, the animal being easily startled, with fixity of the eyes, staring at some imaginary object, the pupil being the while dilated, and the ears moved backwards and forwards, as in a blind horse, when it hears some strange sounds. At one time, during the day and night upon which it was ill, it seemed to present some signs similar to those of laminitis, shifting its weight from one foot to another, and swinging its body backwards. At the same time we noticed convulsive twitchings of the superficial muscles, difficulty in swallowing, spasm of the throat, a hoarse cough, acceleration of the breathing, a quickened pulse, and a flow of saliva from the mouth. After a while it became very furious, fought violently, destroying everything within its reach. Gradually signs of paralysis of the posterior extremities became apparent, and at last it was unable to rise. It would, however, make violent efforts to do so, and during a rapid spring or jump one thigh was fractured. The efforts to rise were not discontinued even after this accident, and so violent did it become, and so strenuous were its efforts to get at something, that the other thigh was broken. It still continued to sit upon its haunches, and to fight with its fore feet, all the while tearing at its breast with its teeth. Seeing that both tibiæ were fractured, the owner consented to what had long before been urged upon him—to have the horse shot.

In the stallion and mare it is stated that the sexual desire is

augmented; that the stallion has frequent erections, and neighs in a harsh tone, and the mare stands with her hind legs apart, showing signs of œstrum.

The remissions and paroxysms are less apparent in the horse than in the dog. In the latter, the animal seems for a time to be almost free from the disease, but in the horse the absence of anxiety, restlessness, and exhaustion is of much shorter duration, and the fits of violence more violent and prolonged, so much so, that it soon becomes prostrated, and dies in frightful convulsions upon the second, third, or fourth day.

Hydrophobia in man is generally characterised by a sensation of intense pain in the seat of the wound, the cicatrix becoming red and irritable, the pain shooting from the extremities (if the wound be situated upon them) to the trunk. It seems most probable that this sense of pain is felt in some instances by the horse, and is manifested by the persistent efforts to bite the seat of the wound, particularly, as in the case described, if it be within reach of the teeth.

The symptoms of rabies in the cow, sheep, goat, &c., are very similar to those in the horse; in all there is "loss or depravity of the appetite, prostration, great restlessness, increased excitability, muscular tremblings, a flow of saliva from the mouth, excitation of the sexual desire, especially in the bull, difficulty in swallowing, and manifestations of hallucinations as well as disagreeable sensations in the seat of the wound."—(FLEMING.) And succeeding to these signs are those of great violence, paralysis of the hind extremities, emaciation, and finally coma and death.

Medical treatment, after rabies has established itself, is of no avail.

#### PREVENTIVE TREATMENT.

Complete excision of the bitten surface, as soon as possible after the infliction of the injury, is the best that can be adopted.

If the wound be superficial, the free application of the caustic potash (*Potassa fusa*) or of the nitrate of silver is sufficient; but if deep or much lacerated, the parts are to be carefully excised before the caustic is applied. Where this has been done in man seven out of ten escape, whilst if no such means are used

eight out of ten die.—(BOLLINGER.) It is very difficult in the lower animals, covered as they are with hair, to know whether the part bitten be confined to any particular region of the body, no matter how carefully the skin may be searched. It is therefore advisable to have the bitten animal shaved as quickly as possible, in order that all scratches be brought into view, and that they may be dressed with the caustic. In dogs preventive treatment should not be resorted to, but the bitten animal ought at once to be destroyed. It is very true that all dogs inoculated with the rabies poison do not become mad; the risk of their becoming so is, however, so great, and the mischief they might inflict is so grave and important in its nature, that the chance of their doing such mischief should never be allowed. Whilst the above precautions are considered necessary, it cannot be said that they are always effective in preventing the disease in bitten persons or animals. Pasteur, however, as is well known, had carried out his system of vaccination by attenuated virus to almost a universally successful conclusion, and he anticipated that, although the time was still distant when canine madness would be extinguished by vaccination, he hoped, pending that consummation, that he would be able to avert the consequences of a bite from a mad dog. He said—"Thanks to the duration of incubation after a bite, I have every reason to believe that patients can be rendered insusceptible before the mortal malady has had time to declare itself."

M. Pasteur's anticipations were put to the test by a Commission appointed by the Minister of Public Instruction, consisting of MM. Bouley, President; Paul Bert, Bécand, Vulpian, Tisserand, Director of Agriculture, and Professor Villemin.

Forty dogs were submitted to the bites of rabid dogs. Twenty of these healthy dogs had been previously inoculated with attenuated virus, whilst the other twenty were not inoculated; the rabies showed itself exclusively among the non-inoculated dogs.

M. Pasteur was requested by the Commission to perform his proof experiments in a more expeditious and certain manner than by only submitting healthy dogs to the bites of rabid ones. He having found, first, that if nervous matter from the bulb of the brain of a rabid animal be inserted on the brain—by trephining—of a healthy dog; second, that when the virus was injected into the veins; and third, that when the dogs were

repeatedly bitten about the head by rabid ones, a rapid and fatal rabies occurred within twenty days—therefore proceeded to carry out a series of experiments on protected and non-protected dogs, with the result that none of the protected animals had, up to 10th July, 1884, shown any signs of the disease, whilst twelve of the unprotected ones, including two which were repeatedly bitten about the head, had succumbed to the disease.

The dogs still remaining healthy were to be watched by veterinary surgeons for a year, to see whether the inoculations hold good permanently or only temporarily. These experiments proved entirely successful, and, acting upon this conclusion, Pasteur continued to apply preventive inoculation to man, and with remarkable success.

#### PATHOLOGICAL ANATOMY.

From the symptoms observed during life, the conclusion naturally suggests itself that the brain and its membranes are the seat of organic lesions. Indeed, the specific action of the poison appears to be exercised, particularly in the first instance, upon the medulla oblongata and the par vagum, the branches of which seem to lose their natural properties; hence the difficulty in swallowing, the depraved appetite, alteration of the voice—or its entire loss in the dumb form—as well as the convulsions of the respiratory muscles, are all due to derangement of this nerve; and as the nervous system of the animal becomes more and more deranged, complete paralysis of the respiratory muscles occurs, and the animal dies from asphyxia.

The principal *post mortem* appearances are œdema or congestion, sometimes in patches, of the brain and spinal cord, particularly at the base and *plexus choroides*, effusion into the arachnoideal space, cerebral ventricles, and the cerebro-spinal substance, and softening of the membranes. On the lower surface of the *medulla oblongata*, at the origin of the seventh, eighth, and ninth pair of nerves, the membranes are generally highly injected, thickened, softened, and matted together. The liver, kidneys, spleen, and the muscular system are congested. The bladder is empty, and its mucous membrane covered with petechiæ. The lungs are greatly engorged with blood. The blood in the vessels is but imperfectly coagu-

lated, often black and tarry, sometimes bright and red, in appearance. The mucous membranes of the pharynx, oesophagus, stomach, and bowels are either greatly congested, with extravasation of blood on their surface, or diffusely inflamed. Patches of extravasation are particularly met with on the gastric mucous membrane, and account for the hæmorrhagic vomiting which is sometimes witnessed during the illness. The contents of the stomach are generally of a peculiar nature, consisting of hay, straw, stones—in fact, of a collection of the most incongruous materials, which, owing to depraved appetite, the animal has picked up during life. This appearance is of great value, as it proves most conclusively that the dog has died rabid.

The tongue is often wounded by the teeth; its papillæ congested; and the salivary glands enlarged and vascular. In “dumb madness” the congestions, more particularly those of the upper part of the respiratory and digestive tracks, are developed to a greater extent than in the other form of the disease.

#### DIAGNOSIS OF RABIES.

“In a case of suspected rabies in a dog, the animal should not be killed immediately, but should be kept under observation for three or four weeks and then killed.

“Moderately thin smears on slides are made from (a) the cortex in the region of the fissure of Rolando (the crucial sulcus in the dog), (b) the hippocampus major, (c) the cerebellum. They are dried in the air, fixed for five minutes in methyl alcohol, and then stained in weak Giemsa (1 drop stain, 1 c.c. distilled water, with 1 drop of 1 per cent. potassium carbonate solution to every 10 c.c. of the dilute stain) for three hours. The stained films are then washed in running tap-water for one to three minutes, dried with filter-paper, and examined for the Negri bodies.

“Or the moist films may be fixed in methyl alcohol, and, without drying, stained for one minute in a mixture of 10 c.c. distilled water, 3 drops of a saturated alcoholic solution of basic fuchsin, and 2 c.c. of Löffler's methylene blue. Eosin-methylene blue mixtures may also be used.

“The cytoplasm of the bodies stains orange, pink, red, or magenta, the central nuclei are granular and appear bluish or purplish.”—HEWLETT: *Manual of Bacteriology* (J. and A. Churchill).

## CHAPTER XXIII.

### CONTAGIOUS DISEASES—*continued*.

#### GLANDERS AND FARCY (EQUINA).

*Definition.*—A malignant, contagious, and fatal disease, due to the introduction into the animal economy of a virus (said by Dr. Strück of Berlin to consist of an organism—the *Bacillus mallei*—about the same size as that of tuberculosis), which, infecting the whole system, shows specific effects more especially upon the Schneiderian mucous membrane, the lungs, and upon the lymphatic glands and ducts. Glanders, and its variety farcy, are capable of transmission to man, in whom the virus seems to increase in malignancy, ass, mule, sheep, goats, dogs, the feline species, and even to mice and rabbits. Cattle, pigs, and fowls resist the action of the contagium, even when inoculated.

#### HISTORY.

Glanders was described by Aristotle,<sup>1</sup> by Vegetius,<sup>2</sup> and other early writers, under the terms morbus humidus, cy-moira, capitis morbus, &c.; and its variety farcy, as morbus farcimosis, vermis equi, vermis volaticus, farcina equi, &c.

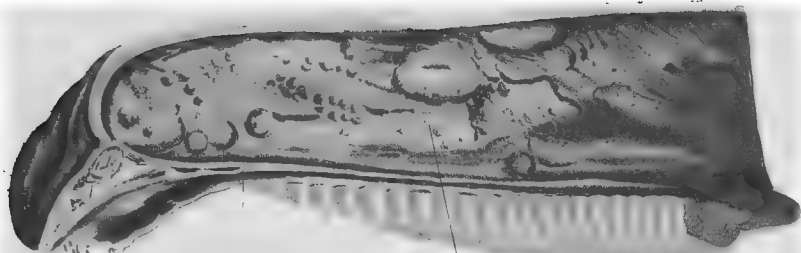
Glanders is a disease of all climates. The contagious nature of glanders was for a long time a disputed point; it is, however, now determined beyond question that its *propagation* is by contagion and infection only.

Glanders may occur under at least four forms, namely, acute glanders, chronic glanders, acute farcy, called by some bud farcy, and chronic or button farcy.

<sup>1</sup> Aristotle, *De Hist. Anim.*, lib. viii. c. xxv.

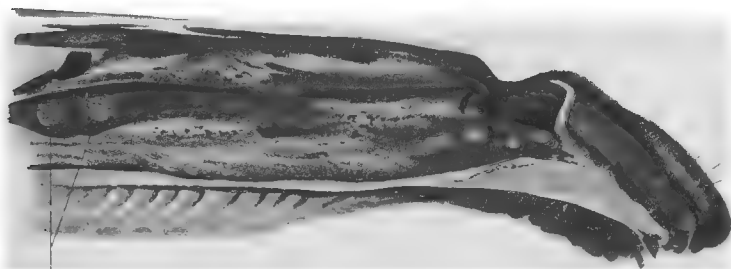
<sup>2</sup> Vegetii, *Renati Artis Veterin.*, lib. i. c. vii.

## PLATE X.



Lead-coloured  
Mucous Membrane.

Confluent Ulcers.



Ulcers.

### GLANDERS AFFECTING NOSTRILS OF HORSE.





## CAUSES.

It is now believed that equina originates from contagion only. Many debilitating influences act as predisposing causes, such as old age, bad food, overwork, exhausting diseases, and general bad management; specific miasmatic or animal poisons, such as those generated in localities when large numbers of horses are congregated together, in camps, barracks, large cab or other establishments, even when the stables are well ventilated, lighted, drained, and the animals well attended to in every way, but more particularly where the stables are ill-ventilated, badly drained, dark, and foul. Horses, when crowded on board ship, are very liable to this affection, and the Arabs in transporting their horses from Arabia to India always choose that part of the year when the passage is shortest, lest the accidents incident to a long voyage might oblige the hatches to be closed, and want of ventilation facilitate the spread of glanders. I have also observed that glanders is developed in new stables, where the walls are not thoroughly dry—where in fact, in common language, they are said “to sweat”; and, finally, glanders occurs as a sequence to exhausting diseases, more especially if the animal be old, or of a bad constitution. These causes, and a generally vitiated condition of the animal system, may be said to predispose glanders—(1.) By causing the introduction into the blood of vitiated or decomposing material generated in the external surroundings of the animal; (2.) By inducing the formation of degenerated material within the animal system; (3.) By preventing the excretion of the degraded constituents normally generated within it by natural tissue changes, or excessively formed within it by various disordered functions, or introduced into it from without.

The most common forerunner of glanders, more particularly of that form of it known as farcy, is the disease commonly called diabetes insipidus or polyuria. It cannot be said that in diabetes there is any obstruction to the excretion of degraded tissue; indeed, the reverse is the case, excretion of urine being enormously increased. If we look deeper into the matter we shall, however, see that the polyuria is associated with rapid tissue changes, rapid emaciation of the body being a most prominent

symptom, with debility arising from degradation of tissue, and from the presence of the degraded materials within the circulatory fluid. So apparent is this condition, that it has been truly said that diabetes, when arising from no cognizable cause, is often indicative of a general breaking-up of the constitution.

Although glanders and farcy are one and the same disease, differently manifested, farcy is apparently more frequently developed spontaneously than glanders. This circumstance, and the possibility of recovery from mild attacks of farcy, has led some to conclude that they are two separate disease; but direct experiment has proved that the virus is identical in both forms of the disease. The discharge from the nose of a glandered horse, when introduced into the systems of other horses, may in one produce glanders and in another farcy, whilst the pus from a farcy ulcer may produce in the inoculated animal glanders, farcy, or both; and the common termination of farcy, if a horse affected by it be allowed to live a sufficient length of time, is glanders, and of glanders farcy. Such an animal is described as being "both glandered and farcied."

#### CONTAGION.

Glanders, and its variety farcy, are highly contagious and infectious; and when once introduced into a stable are almost certain to spread amongst the horses there located.

As already stated, the contagium is found to be an aërobic bacillus discovered by Loeffler and Schütz, 1882, called the *Bacillus mallei*. These organisms are seen in the form of minute rods  $\frac{1}{22700}$  long, and from  $\frac{1}{60800}$  to  $\frac{1}{20320}$  of an inch broad; they are rather thicker than the tubercle bacillus, and retain the methylene blue stain. They are found in the nodules in the lungs, liver, Schneiderian membrane, and other involved structures, and are free or within the cells of the part. They are non-motile, straight, or slightly curved, with rounded ends, and sometimes appearing as diplo-bacilli.

They stain readily with aniline dyes, but do not retain their colour with Gram's method. The best stain is that of Schütz: potash solution, 1 in 10,000, and concentrated alcoholic methylene blue, equal parts. Wash the section in a

watch-glass full of water, containing four drops acetic acid. Transfer for five minutes to 50 per cent. solution of alcohol, fifteen minutes to absolute alcohol, clarify in clove oil, and mount in Canada balsam.

By careful staining, the glanders rods may show what is known as mitachromatic staining; that is, comparatively clear spaces may be seen, as is always the case in staining tubercle bacilli with carbol fuchsin, by Ziehl Neilsen's method. It is unknown what these spaces are, but it is probably some fatty

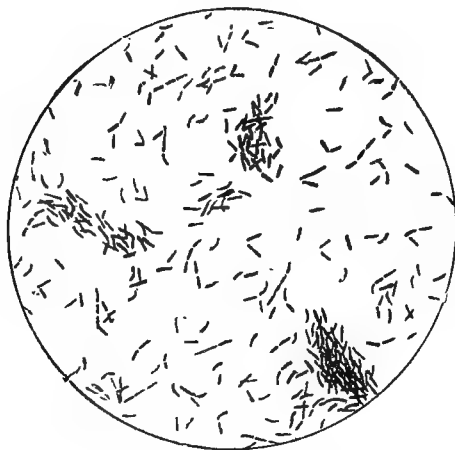


FIG. 17.—Glanders Bacilli.

substances that resist the action of the stain, as they are decidedly not spores.

The specific poison can be introduced into the system either by the skin or mucous membranes. By inserting the virus under the skin with the point of a lancet, by rubbing the greasy heel of a horse, and by inoculating the mucous membrane of the nose, the disease has been produced.

With regard to the transmission of the *contagium* of glanders otherwise than by actual contact, opinion seems to be divided, some writers maintaining (and the experiments brought to bear upon their conclusions are formidable) that it is impossible to communicate it in any one of its forms by compelling healthy

animals to inhale the expired air of those suffering from the disease, whilst others maintain that it is both contagious and infectious. The first opinion, if acted upon in every day practice, would, I opine, lead to disastrous results, and should not be entertained; for even if we were to grant that the virus is contained in the discharges from the ulcers, numerous experiments have proved that pus and other organised cells float in the atmosphere; the specific virus contained in these cells may thus be easily conveyed from one animal to another. Again, it must not be forgotten that in some instances glanders may exist without any external manifestations, *i.e.*, without discharge from the nose, nor from farcy ulcers, but even in this form it is capable of propagation.

Farcied matter, made into balls, and introduced into the stomach of a horse, has caused glanders; and whichever way the virus has been introduced, once absorbed, it infects the whole blood, as has been proved by the experiment of the late Professor Coleman, who says—"I have produced the disease by first removing the healthy blood from an ass until the animal was nearly exhausted, and then transferring from a glandered horse blood from the carotid artery into the jugular vein of the ass. The disease in the ass was rapid and violent in degree; and from this animal, by inoculation, I produced both glanders and farcy." Professor Coleman also experimented on asses with virus obtained from man. He directed two asses to be inoculated with matter taken from the arm of a man labouring under the malady, which resulted from a puncture received in dissecting a glandered horse, and both asses died of glanders. These experiments have been repeated, and similar results obtained by Girard, Hering, and Leblanc.

It must not be concluded from these facts that glanders is a septicæmia. It is conceivable that, when an animal is infected, the lesions set up will tend to break down, and thus the organisms being liberated may gain entrance to the blood-stream. They will then be carried by this fluid until they obtain a resting-place, where they will locate and set up fresh lesions. It is in this way that most probably a miliary condition is set up in the lungs, spleen, &c., as is seen also in tuberculosis.

*Period of Incubation.*—Like all morbid poisons, that of glan-

ders has its period of latency, which is, however, generally short. In the ass, the submaxillary glands become swollen and tender the second or third day after inoculation, and a discharge from the nostril occurs from the third to the sixth day. In some instances the incubation is much longer—from one to three or even six weeks; one case is mentioned in the *Procès-verbal de l'École de Lyons* where the disease did not appear till the end of the third month after inoculation. The matter in this instance was obtained from a farcy ulcer, and the disease appeared at the precise point of the inoculating puncture.

#### ACUTE GLANDERS.

*Symptoms.*—The disease, preceded by increased temperature of the body, appears suddenly, ushered in by rigors, sometimes of a most persistent character. In one case which fell under my notice the rigors continued without intermission for three days and nights, at the end of which time a sanguineous discharge issued from both nostrils, the Schneiderian mucous membrane became violently inflamed and deeply ulcerated. More commonly, however, the rigors are not so persistent, but they are always more or less observable; the temperature is sometimes as high as 106° or even 109° F., the breathing is accelerated, the pulse feeble, rapid, and even dicrotonous, the heart's action palpitating and accompanied by metallic tinkling, the appetite fails, the pituitary membrane, at first of a dark copper colour, with patches of ecchymosis of a dark red hue, becomes purple, and the patches are rapidly converted into pit-like, ragged-edged ulcers, from which issues a copious sanious discharge. The submaxillary lymphatic glands enlarge. Other lymphatic glands inflame, enlarge, suppurate, burst, and expose raw, unhealthy-looking sores, from which issues a more or less ichorous and irritating purulent material. The eyes are weak, and a discharge issues from them; the nostrils are often swollen. The breathing is hurried, irregular, and impeded by the swelling of the nostrils, and by the glutinous discharge drying around them; abscesses speedily form along the course of the lymphatics of the face. The urine is pale, watery, and increased in quantity.

Acute glanders is rapidly fatal, and the *post mortem* exami-

nation reveals the following lesions:—The mucous membrane lining the nasal passage and facial sinuses is violently inflamed, and covered with a numerous crop of pustules and ulcers, from which a purulent or sanious fluid is freely discharged. In many instances, and where the disease has been *very* rapid, the inflammation is diffused over the whole surface of the mucous membrane, which then presents one uniform purple or purplish-black hue, its structure being exceedingly soft and friable from degeneration, and it is easily detached from the bones. Now and then it will be found that the nasal bones, the *septum nasi*, the turbinated and ethmoidal bones, are in a state of necrosis, their surfaces bathed with a purulent discharge, and wholly separated from the mucous membrane. The intermaxillary glands are congested and surrounded by a yellowish exudate, and the lymphatic ducts leading from them are thickened and congested.

The lungs in almost every case of glanders, acute and chronic, are more or less inflamed. Now and then the inflammation is diffuse, embracing perhaps the whole of one or a part of both lungs, whilst in others it is limited to some of the lobes, and presenting the appearance of a series of tumours and patches of congestion of different sizes, and in varying stages of development, scattered throughout the lung tissue. Many of these inflamed spots will be found to contain pus; hence they have been termed tubercles, and the nature of the disease has, from this supposition, been laid down as tubercular; and so close is the relationship between tubercle and glanders, that the editor of the *British and Foreign Medico-Chirurgical Review* says:—"It is in *glanders* that Villemin thinks he has found the closest marks of analogy with tubercle, not only in its anatomy, but also in its symptoms and causation. He seems to have been conducted from the study of glanders direct to the inoculation of tubercle. The characteristic lesion of glanders is a small nodule, which is strewn either in the mucous membrane of the nasal passages, or in the lungs, or, more rarely, in the liver and spleen. At first a greyish-white, firm granulation, composed of cells and nuclei apparently developed by hyperplasia of connective tissue, it soon tends to soften centrally and form ulcers on the mucous membrane, cavities in the lungs. Like miliary tubercle, it occurs isolated or in clusters. Together with this little granulation, streaks and bands of fibrous tissue, as well patches of cheesy

infiltration, are not infrequently met with in the lungs of glandered horses. It is interesting, too, that the same doubts have been raised concerning the real nature of these 'infiltrations' in glanders as in tubercle. They are regarded by Villemin as one form of glanders, just as in man they are one form of tubercle. As to which is the part primarily affected in glanders—the nasal membrane or the lungs—there is some difference of opinion; Virchow maintaining that the deposits in the lungs are always secondary and by metastasis from the nasal membrane; Phillippe and Bouley being convinced by repeated *post mortem* examinations that the primary lesions are always in the viscera, more particularly the lungs, and that the formations in the nasal membrane are invariably secondary. If, say they, a horse has the 'jetage' (discharge from the nose), it is already thoroughly glandered. It really matters very little which part of the body is first affected. In either case the analogy with a tubercular outbreak remains as strong as can be. The intestinal ulceration of tuberculosis—in which we see the counterpart of the nasal ulceration in glanders—is more often secondary to the pulmonary disease, but occasionally shows itself before any evidence of mischief can be detected in the lungs. Again, glandular enlargement of a severe and persistent kind constitutes an important part of glanders, as it does of tubercle. The mode of invasion is likewise identical in the two diseases; now acute, foudroyant, destroying life in a few days as by an overwhelming blood poison; now chronic, so as to last for years. Further, in the chronic form, the same recurrence of acute attacks complicating and adding to the chronic mischief is observed in glanders as in tuberculosis. To read a description of chronic glanders is, *mutatis mutandis*, to read an account of chronic phthisis. It is, therefore, not surprising that Dupuy goes so far as to say that glanders is a tubercular disease in the horse. In speaking of the supposed causes of tubercle, we purpose presently to follow out still further this remarkable thread of resemblance; but for the present it will suffice to say that glanders is transmissible by inoculation, and contagious from horse to horse, and that it is also unmistakeably communicable from horse to man. Can we hesitate to believe, says Villemin, that the parallel between tubercle and glanders must here find its completion? To conclude, glanders and tubercle are so closely akin that they must

be looked upon as nearly related species of the same genus.”—(BRAITHWAITE's *Retrospect of Medicine*, vol. lviii.)

Whilst admitting the close resemblance between glanders and tuberculosis, and of the similarity of the microbes found in both diseases, we cannot admit their identity, for the broad fact remains that inoculation with glanders produces glanders nodules, whilst inoculation with tubercle is followed by the development of tubercular tubercles.

The essential difference histologically between the glanders nodule and a tubercle of tuberculosis is, that in the former it is always distinctly hæmorrhagic, in the latter this is not so. The former tends but slowly to caseation, and seldom to calcification; in tubercle this is the normal and rapid sequence.

The form of pneumonia seen in glanders is very characteristic, the inflamed part resembling an infiltration, with thrombi in the blood-vessels—gangrenous, emphysematous—of a greenish-black colour, and rapidly decomposing after death. The line of termination or demarcation between the healthy and inflamed lung tissue is often abrupt and very distinct. The tracheal and bronchial mucous membrane is more or less highly inflamed; in some instances covered with petechial spots or deeply ulcerated, and thickly covered with an unhealthy discharge, which exhales a gangrenous odour.

The changes which occur in the lymphatic glands in glanders are characteristic. The glands, irritated by the specific poison, become congested and enlarged; their cellular elements proliferate more or less rapidly, and are mixed with a citron-coloured exudate, which invades the surrounding connective tissue. In a few days the glands become dense and hard to the touch, more or less lumpy on their surface, and those in the submaxillary space fixed to the jaw by the now inflamed and indurated vessels which enter their deeper seated parts.

#### CHRONIC GLANDERS.

*Symptoms.*—In some instances the disease presents itself in such a mild form that the general health is scarcely affected. There will be a discharge from one or both nostrils, generally from one nostril, and that very often the near (left) one. The submaxillary



lymphatic glands are swollen and hard; the hardness and swelling are of a remitting nature, very often varying in size in a short period. For example, a horse may be left at night with scarcely any discoverable swelling, and found in the morning with a hard knot under the jaw, which is both easily seen and felt. The swelling may continue for several days, afterwards slowly disappear, and then reappear as rapidly as before. This condition may exist before any discharge issues from the nose, and a horse so affected is elegantly said to be "jugged." If the nostril of such a horse be examined, it will be found to be paler in colour than natural, or perhaps tawny, coppery, and sometimes of a dull leaden hue. The discharge of glanders presents a starchy or glue-like appearance, adheres to the nostrils, where it dries and accumulates, causing the nasal opening of the affected side to appear smaller or more contracted than in health.

These appearances, in addition to a weak or debilitated condition of the eye of the affected side, may be all the symptoms present in a case of chronic glanders; indeed in some instances there may be nothing but the discharge from the nostril to lead the practitioner to suspect anything wrong with the animal, and the diagnosis is consequently very difficult, more particularly if the case is a solitary one; but where glanders is found to exist in a stud of horses, any suspicious symptom becomes significant. I have said nothing about the glanders-ulcer, because in many instances of chronic glanders the ulcer is undiscoverable; indeed in some rare cases ulcers are never found either before or after death. For this reason Percivall limited the term chronic to that form in which no ulcers could be detected. He says, however, that they are always present in the frontal sinuses.

Before describing the more common form of glanders—namely, that in which ulceration is a characteristic sign, and called by Percivall "subacute glanders"—I shall call the reader's attention to what may be termed a spurious form of glanders; that is to say, a form of glanders where there is neither discharge from the nose, ulceration of the Schneiderian membrane, nor enlargement of the submaxillary glands; but in which all these may become apparent if the animal live long enough. This form may be appropriately termed *pulmonary glanders*, and the symptoms are as follows:—The animal is languid, unthrifty, loses flesh, sweats on the slightest exertion; the visible mucous membranes are

pale; diarrhœa is easily induced, and there are occasional attacks of diabetes. There is a dry, dull, soft cough; the appetite is very capricious; the hair is easily removed from the mane and tail; the legs are sometimes very fine, sometimes more or less œdematous. The affected animal may remain in this unsatisfactory condition for several weeks, or even months, and may die from marasmus and debility without any other symptoms becoming apparent, or all the signs of glanders and farcy may very rapidly develop themselves, and carry it off in a few days. If a case of this kind occur in the horse first attacked in a stud, or if in a horse where no others are kept, it is now possible to arrive at a satisfactory conclusion as to the nature of the malady by inoculating the animal with mallein, hereafter referred to.

The *post mortem* examination of a horse which has presented these symptoms prior to death will reveal the lungs studded with nodules, varying in size from a pin's head to a pigeon's egg, or even larger. Some of these nodules will be found to consist of an organised exudate—lymphoma—of a pearly grey appearance, and rather hard; others will contain pus enclosed in sacs—*vomicæ*; whilst others will be found in a degenerated condition, and presenting the appearance of rotten cheese, the degraded material consisting either of inspissated pus or a caseous metamorphosis of the exudate, in which the bacilli may be discovered by microscopical examination. In many instances the trachea is found studded with ulcers, and some of the deep-seated lymphatic glands will be found enlarged, or in a suppurating condition. One remarkable pathological change which I have seldom failed to observe either in this or any but the most acute form of glanders, the splenic veins are filled with thrombi; the larger branches in particular being generally obliterated by hardened fibrinous coagula.

Glanders, as most commonly met with in this country, presents the following signs:—The horse is generally more or less off its feed, has a tendency to shiver on the slightest cold; its coat is rough and unhealthy, "has lost the bloom of health;" it may or may not cough; the appetite is capricious; and perspiration is induced by slight exertion. There is a discharge of a starchy or gluey material from one or both nostrils; the discharge is often tinged with blood. In some instances

recurrent hæmorrhage from the nostril is a premonitory sign of glanders. The mucous membranes are pale and unhealthy, and that covering the nasal chamber, from which the discharge issues, is studded over with deep, pit-like ulcers. The ulcers are characteristic, being excavated, as if cut with a punch, but after a time they become ragged at their edges, irregular, enlarged in all directions, and confluent. The spaces between



FIG. 18.—Mallein reaction, showing swelling in neck.

the ulcers are covered with hard, yellowish pimples, which soon ulcerate. The eye of the affected side is weak, and looks smaller than its fellow, and an unhealthy discharge often issues from it over the face. The submaxillary lymphatics of the same side enlarge and form a tumour—sometimes single, sometimes lobulated—which is more or less firmly adherent to the surrounding tissues. This tumour seldom suppurates; occasionally, however, I have seen it developed into an abscess,

which, after having discharged a little unhealthy pus, has healed slowly, the tumour, however, continuing as large, or even larger, than before the suppuration.

If the animal is allowed to live, these symptoms remit, and some of the ulcers may cicatrise, but they finally increase in intensity, and upon the application of any slight cause of disorder become rapidly developed into those of acute glanders. If the horse is well taken care of, it may do moderate work for a long time whilst affected with chronic glanders. In the course of time, however, symptoms of farcy appear; it then becomes a loathsome object, and if not destroyed, acute glanders sets in and carries it off in a very short time.<sup>1</sup>

Chronic glanders is more frequently seen than the acute form in the horse, but in the ass and mule the latter is more frequently met with.

In acute glanders the period of incubation is very short—from three to seven days; whilst in the chronic form the period is very uncertain, extending from a few days to several months before actual clinical symptoms become apparent.

It is practically certain, however, that the incubation period does not extend beyond twenty-eight days. The administration of mallein will always with certainty give a reaction if there be any disease present, although there may be nothing appreciable to an ordinary clinical examination.

#### ACUTE FARCY.

This form of the disease occurs either as the result of direct inoculation with the discharge of glanders or farcy; from infection through the medium of the air; as a sequel to some exhausting disease, particularly diabetes; from old age and other debilitating influences.

*Symptoms.*—The primary signs are those of fever, elevation of the animal heat to 106° or even to 108° F., rigors, loss of appetite, and swelling of the extremities.

The swellings of acute farcy, commonly confined to the extremities, manifest themselves by engorgement of a whole limb, resembling the swellings of acute lymphangitis or œdema, but

<sup>1</sup> For the differential symptoms of glanders and other diseases accompanied by a nasal discharge, see *Principles and Practice of Veterinary Surgery*, page 493.

PLATE XI.



FARCY BUDS IN VARIOUS STAGES.



presenting an uneven surface, increasing and decreasing suddenly, and attended with pain and lameness. When the engorgement of the areolar tissue diminishes, enlarged lymphatic glands and vessels will then be detected, forming buds and cords. The swellings, which are due to inflammation of the ducts and valves, point and burst, giving exit to a thin, purulent, yellowish material, which generally soon dries, and forms a yellow crust on the surface of the ulcer. These buds are generally found in groups, and away from the articulations.

In some instances farcy manifests itself by, first, the formation of a single painful swelling, which runs on to suppuration, on the flexor tendons of a limb, below the hock or knee, and from which enlarged lymphatic vessels may after a time be traced. Other swellings form on various parts of the affected limb, with intervening enlarged lymphatic vessels, which ultimately burst and present ragged-edged, confluent, unhealthy-looking sores that discharge copiously.

In other instances, farcy is preceded by symptoms of rheumatism in some part of the body. Sometimes the muscles of the neck become acutely painful, and very often, but not always, swollen. The pain and swelling may disappear as suddenly as they came, and appear in some other part of the body, and this may occur repeatedly before any diagnostic signs of farcy manifest themselves; or the seat of pain may be the muscles of the chest, simulating pleurodynia or pleurisy, the animal groaning if made to move sharply or suddenly, and with a catch in his breathing diagnostic of thoracic pain. Again, one limb may suddenly become swollen and painful, causing much lameness. The tumefaction, heat, and lameness may recede, and attack the other limb; and such attacks may appear repeatedly and for several months, the animal's general health during the remissions being scarcely affected; but at length the disease assumes a more marked character, and unmistakeable symptoms of farcy appear, or glanders results, unpreceded by any true signs of farcy. In other instances, extreme lameness may appear in a limb without any traceable cause, and continue for days, or even weeks, without inducing any observable local change; generally, however, farcinous engorgement succeeds, and relieves excessive pain, and as a rule extreme pain is not of long duration. But it may be safely stated that equina is manifested in such a variety of ways as sometimes to mislead the most experienced.

## CHRONIC FARCY.

In chronic farcy the local symptoms generally precede any apparent febrile disturbance; but if the thermometer be employed for the examination of all horses in a stud where glanders exists, some elevation of the temperature of the body may be apparent in the infected prior to the development of any local symptoms.

These local symptoms consist of circumscribed inflammatory swellings, running in the direction of the principal vessels, which suppurate and burst, without much accompanying engorgement of the surrounding areolar tissue.

The circumscribed elevations or buds are connected together by corded lymphatic vessels, and wherever a valve is situated in a lymphatic duct, there a swelling will usually appear, and a bud will form. The buds are ranged in groups about the inner and outer aspects of the thigh, fore arm, flank, neck, and head. From the circumstance that the enlarged cords and buds run in the same direction as the veins, the old farriers concluded that farcy was a disease of the veins; dissections, however, soon dispelled this delusion, and the reason why the disease accompanies the vessels is explained by the fact that the lymphatics and blood-vessels run in company.

In some instances farcy is found confined to the cervical lymphatics. An examination of the neck along the course of the jugular vein will enable the veterinarian to detect the lymphatic duct swollen, hard, and presenting irregular knots along its course. Suppuration seldom occurs, but the animal sooner or later presents signs of glanders or of farcy in some other part of the body.

Chronic farcy differs from the acute only in intensity and duration, and is the only form of equina which is at all amenable to treatment.

The Contagious Diseases (Animals) Act provides, however, for the destruction of glandered and farcied horses. I think that the provisions of the Act should be strictly carried out, and compensation paid from the imperial exchequer, as the disease, in whatever form it appears, has hitherto remained incurable, and is always a source of danger both to human and



animal life. It is stated, however, that cures have been effected by fourteen injections in forty days of mallein from a cat inoculated with glanders. Decroix and Bougome, two Algerian veterinary surgeons, are stated by Semner to have cured farcy by cauterisation and extirpation of the farcy buds—an exploded practice found to be useless in this country long ago. Brusaco, of Turin, and his pupils are stated to have cured 50 per cent. of glandered horses with carbolic acid, iodine, sublimate and sulphate of iron; whilst others claimed to have cured horses with tracheal injections of iodine and iodide of potassium; and in France they are satisfied with the curative effects of creosote in oil injected hypodermically. Semner again mentions that ox blood serum is useful in the cure of glanders. Pilarios states that he cured eight cases in this stage of the disease by repeated weak injections of mallein.

Glanders in man has been cured by frequent injections of mallein. Semner, however, prefers ox serum to mallein, and says that it has a more marked effect in destroying the virulence of the bacilli than mallein. Like Professor Macqueen, from whose observations (Central Veterinary Medical Society) I am now quoting—"To prevent misunderstanding, I ought to say that a glandered horse is said to be cured when it ceases to react to mallein. So much for the curability of glanders."—(*Veterinary Record*, April 20th, 1895).

I entirely disapprove of the practice of working animals whilst affected with this malady, and would recommend the strictest surveillance by properly qualified inspectors.

#### THE ANTISEPTIC PREVENTIVE TREATMENT OF EQUINA.

When glanders appears in a stable of horses, in addition to the removal of all affected animals, and the inoculation with the mallein test of all in contact, it is most essential to attend carefully to the ventilation, drainage, food and water, and to the cleansing and disinfection of the stables, fittings, harness, and other appurtenances, the removal of contaminated wood-work, the painting of all fixtures, lime-washing, or even scraping and then lime-washing—the wash to contain a pint of crude carbolic acid to every bucketful—and to the prevention of overcrowding, exhaustive work, and all debilitating influences. It has been found serviceable to administer for several weeks to all horses in

the same stud, with every meal, two ounces of the hyposulphite of soda, or two drachms of the chlorate of potash. American practitioners laud the hyposulphite in farcy, and say that the sores require no treatment except cleanliness.

McFadyean says: "Thorough disinfection of infected articles or premises is not difficult. The vitality of the bacillus is not great, being destroyed in a short time by exposure to sunlight, and by such substances as carbolic acid and corrosive sublimate. The bacillus retains its vitality in ordinary clear water for some ten or fifteen days, and silk threads, which had been saturated with pure cultures and then dried, were found infective as long after as eighty days. In ordinary positions, such as are afforded in stables, the bacillus has to contend with other organisms which impair its vitality. The old notions of the necessity for destroying old infected buildings to get rid of infection are entirely wrong. The cases in which glanders has broken out in horses that were put into old stables which had been badly infected months or years previously are explained by the simple fact that some of the animals suffered from latent glanders when placed in the stable."

**THE MALLEIN TEST.**—Following the example of Koch, the discoverer of tuberculin, Helman of St. Petersburg successfully obtained the now recognised test for occult glanders, namely, "mallein," which is a glycerine extract of cultures of the *Bacillus mallei*. This extract is sterilised by heat, and attenuated to ten times its weight with a two per cent. solution of carbolic acid. Thirty minims of this solution injected into a glandered horse causes an elevation of temperature of  $2^{\circ}$  to  $3^{\circ}$  in about nine to fifteen hours. There is also depression and increased rapidity of the pulse.—(See Preparation of Tuberculin.) As a rule, within a few minutes after the injection there is a swelling at the seat of operation, and this swelling may remain in a healthy animal for the matter of twenty-four hours and then disappear, which must not be confounded with the "swelling of reaction."

This latter swelling develops slowly, and is doughy to feel and flat to look at. It shows no sign of "pointing." Within twenty-four hours it may measure 6 in. long by 6 in. broad by 1 to 2 in. thick, and will go on increasing in length and

width, but not in thickness, for even another twenty-four hours, and then measure 10 in. long by 10 in. broad (see Fig. 18, p. 261). After two or three days from inoculation, this swelling, which is extremely painful all the time, will gradually subside.

In healthy animals there is no elevation of temperature or other febrile symptom, nor is there any swelling other than that simply due to the irritation of the injection, and which disappears in the course of twenty hours, or even less.

It must be borne in mind that both mules and horses which are recovering from or have recently recovered from some of the forms of influenza and other bacterial diseases, may show a typical glanders reaction if injected with mallein.

This, therefore, much discounts the value of mallein, and in these cases it will be necessary to resort to the agglutination test; though this is uncertain, for Foulerton has shown that the sera of diphtheria and typhoid also produce agglutination of the bacilli of glanders.

#### STOMATITIS PUSTULOSA CONTAGIOSA OF THE HORSE.

The *Veterinary Journal* for November 1878 contains a report on this disease by Professors Eggeling and Ellenberger of the Berlin Veterinary College, translated from the *Archiv für Wissenschaftliche und Prätische Thierbeilkunde* by G. A. B. I am indebted to Dr. Fleming for the woodcuts illustrating the eruptions characteristic of the disease.

They state that during the summer of 1876 several cases of a contagious disease appeared in and outside the clinic of the Berlin Veterinary College, presenting phenomena similar to those of variola, and chiefly affecting the mucous membrane and external integument.

Some cases outside the clinic had been declared by the inspecting veterinary surgeons to be suffering, or suspected of suffering, from glanders.

Most of the patients were from four to five years old, in good condition, and showing but slight indications of general illness; having on the whole a lively appearance, smooth and glossy coats, and eating bran mash with good appetites; but whilst eating hay, large quantities of saliva flowed from the angles of the mouth.

Some of the horses stood with depressed and stretched-out heads; the temperature of the skin was warmer than natural, although the ears and extremities were cold. The pulse was 60 per minute, and full; the respirations normal. Even those showing the worst symptoms ate their food eagerly, but evinced

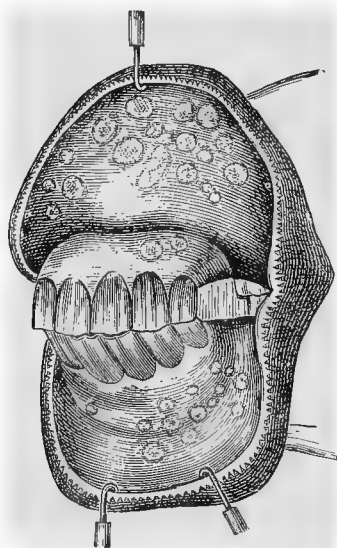


FIG. 19.—Ulceration of dental surface of lips.

pain during prehension, masticated slowly, swallowed with difficulty, and saliva was secreted in abundance, and a fine chlorophyll green discharge flowed from the nasal cavities; when water was taken large quantities of it was returned through the nose. The Schneiderian membrane was of a red rose colour; both the submaxillary glands were enlarged to about the size of a hen's egg—hard and knotty—but unattached to the submaxillary bone or integument. The oral mucous membrane became intensely hot, red, and covered by a tenacious secretion. On passing the hand over it, small firm nodules, varying in size from a millet to a lentil seed, could be felt. These nodules rapidly increased in number and size, extending to the cheeks, tip of tongue, *frænum lingue*, inferior surface of tongue and upper lip, and in one or two days later their summits presented a white appearance, or the epithelium was removed, and a small ulcerated surface was apparent, and in four or five days the ulcerations were numerous, the dental surface (see Fig. 19) of the lips, the tip of the tongue (see Fig. 20), *frænum*, and inferior surface of it, being specially affected.

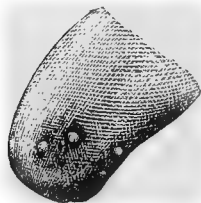


FIG. 20.—Ulceration of tip of tongue.

About the sixth or eighth day the ulcerations began to heal. Four horses condemned as glandered, presented, in addition to the above phenomena, ulceration of the external integument, namely, on the upper lip, cheeks, and anterior extremities.

The ulcerations of the skin in two of these cases were more numerous than in others observed before or since, and the prescapular glands were swollen in one case.

In one case the nodules appeared between the external nares and Schneiderian membrane; in another, on the skin of the breast and fore extremities; in fact, on those parts on which saliva is most likely to be thrown, several nodules about the size of a pea, and two ulcers about the size of a threepenny piece were seen. On no other parts of the body could nodules or ulcers be found.

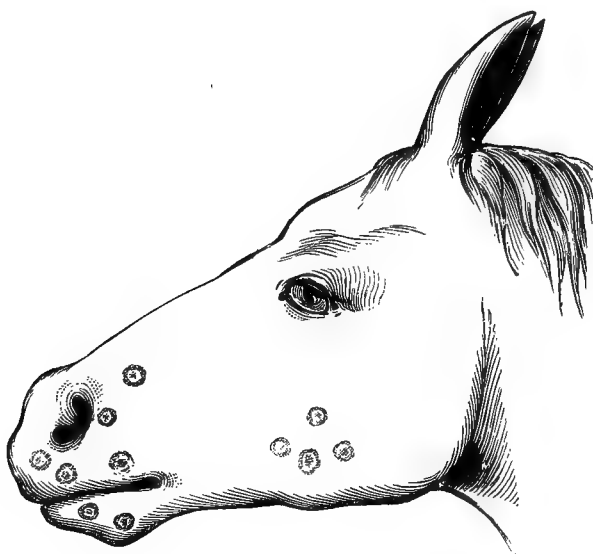


FIG. 21.—Ulceration of skin of lips and cheeks.

The disease runs a rapid course, the small nodules on the mucous membranes soon becoming prominent, presenting a smooth, round, and sharply defined surface, with a yellowish-white summit, or the epithelium becomes removed, and they are transformed into round or slightly angular ulcers, varying in size from a threepenny piece to a shilling—they, in their turn, becoming encrusted, somewhat resembling pus scabs. Microscopically examined, they were seen to consist of pus corpuscles and epithelial cells, with red blood corpuscles. Fungi and micrococci were also present. The ulcers on the legs were generally larger than those on the mucosa.

The healing process commenced in the ulcers about the sixth or eighth days, and all the cases under treatment resulted in the animals getting quite clear of the disease in from twelve to fourteen days.

Experiments as to the transmissibility of the disease by inoculation and infection were made on sixteen horses, two cattle, four sheep, one goat, three swine, two dogs, one rabbit, and five men, with varying results. Four men and a number of horses took the disease by natural contagion, independently of experimental inoculation.

Below are given a few extracts from the diary kept during the course of the experiments.

*22d May.*—A seven months' old foal was successfully inoculated on the dental surface of the lip, the left ear, and the internal surface of the left fore leg; the resulting eruption was extensive and confluent, and the disease protracted, not having completely ceased until the twenty-first day.

*30th May.*—A healthy foal was placed with the above one, and another was inoculated on the vaginal mucous membrane. The inoculated one gave a negative result, but in the healthy foal the result was positive, the disease running its course, and terminating in eleven days.

*31st May.*—Another horse was placed with the last-mentioned foal. On the 2d of June nodules were formed, the disease terminating on the 12th. Others were inoculated with similar results, and the four foals which passed through all the stages of the disease before were again inoculated, but did not become infected a second time. One showed three small nodules, which disappeared without ulcerating in a few days.

*24th May.*—A calf was inoculated on the vulva. No symptoms appeared, and it was again inoculated on the 30th, after which the disease fully established itself, terminating in eight days. Others were inoculated with similar results.

*23d May.*—Sheep were inoculated with negative results. In pigs the result was also negative. In dogs, nodules appeared, but no ulceration. Rabbits were not affected.

*29th May.*—Mr. Häusel (student) inoculated himself. There was redness and unpleasant itching and swelling of the part, the formation of a pustule, over which a yellow-brownish crust formed, which fell off in fourteen days, leaving a deep cicatrix.

Other four gentlemen inoculated themselves without infection.

A groom was infected, the disease running the course above described, he becoming quite convalescent in twelve days.

In contrasting this disease with that of glanders, the authors say—In a superficial examination the malady might be easily mistaken for that of glanders. In favour of this diagnosis we had its contagious character, the feebleness of the discharge from the nostrils, the swelling of the submaxillary and pre-scapular glands, accompanied with the formation of ulcers in the external integument and Schneiderian membrane (the last in one instance only). Close examination and observation, however, brought to light important differences between this disease and glanders. The most striking deviation was the appearance of the ulcer itself, which presented quite a different character to that of glanders. In these instances the ulcers were round, with sharply defined borders, varying in size from a threepenny piece to a shilling, the edges neither being serrated nor swollen as they are in glanders. The ulcerations were isolated, and independent of the course of the lymphatics. For instance, those on the lips and cheeks were irregularly distributed, and covered with a brownish-white scab projecting somewhat above the surrounding skin. When this was removed a white granular ulcerated surface presented itself. The ulcers were generally superficial, and rarely attained any depth, hardly ever reaching the subcutis. After the sixth day they readily healed.

The disease lasted in individual cases from twelve to fourteen days, and ran through a whole stable in about three weeks.

The disease in question differs then from glanders, more particularly in the character and distribution of the ulcers; also in the absence of lymphatic inflammation; in the quickness with which the ulcers healed; in the non-appearance of ulcers in the Schneiderian membrane; further, in the rapid course of the disease, its short incubative period, and the quickness with which it spread from one animal to another.

The virus seems to lose its virulence after passing through the system of several animals. From the scarcity of horses the experimentalists had to use cattle, when by degrees the lymph lost its infectious properties, until it finally ceased altogether. This characteristic separates the disease from variola, which it otherwise closely resembles.

## CHAPTER XXIV.

### CONTAGIOUS DISEASES—*continued.*

#### EPIZOOTIC LYMPHANGITIS.

EPIZOOTIC lymphangitis is a contagious disease affecting equines and bovines, and is due to a specific organism known as the *Saccharomycosis farciminosi* of Rivolta, an oval-shaped body resembling a melon-seed, and having a well-defined contour and a refractile double outline, and measures 3 to 4  $\mu$  in diameter. Its favourite habitat is the subcutaneous lymphatic system, and the disease much resembles chronic farcy.

Cultivation has up to the present not succeeded in any artificial media. It is difficult to stain except by the Claudius method, and when it is stained it loses its characteristic double contour. If possible, a diagnosis should be made from fresh pus examined wet under a cover-glass. It is, however, possible to dry the pus on a slide as a smear, and when an opportunity occurs place a moist cover-glass over it, and examine. A magnification of 800 diameters is sufficient to see the organism distinctly, but 1,000 renders the diagnosis more certain.

#### HISTORY.

It is only of recent years that this disease has attracted much attention. It caused a great deal of loss in India, to which country it probably gained access with some Italian mules. It subsequently caused considerable trouble in South Africa, from which country it was imported to England. It is indigenous along the Mediterranean Littoral, particularly in Italy.



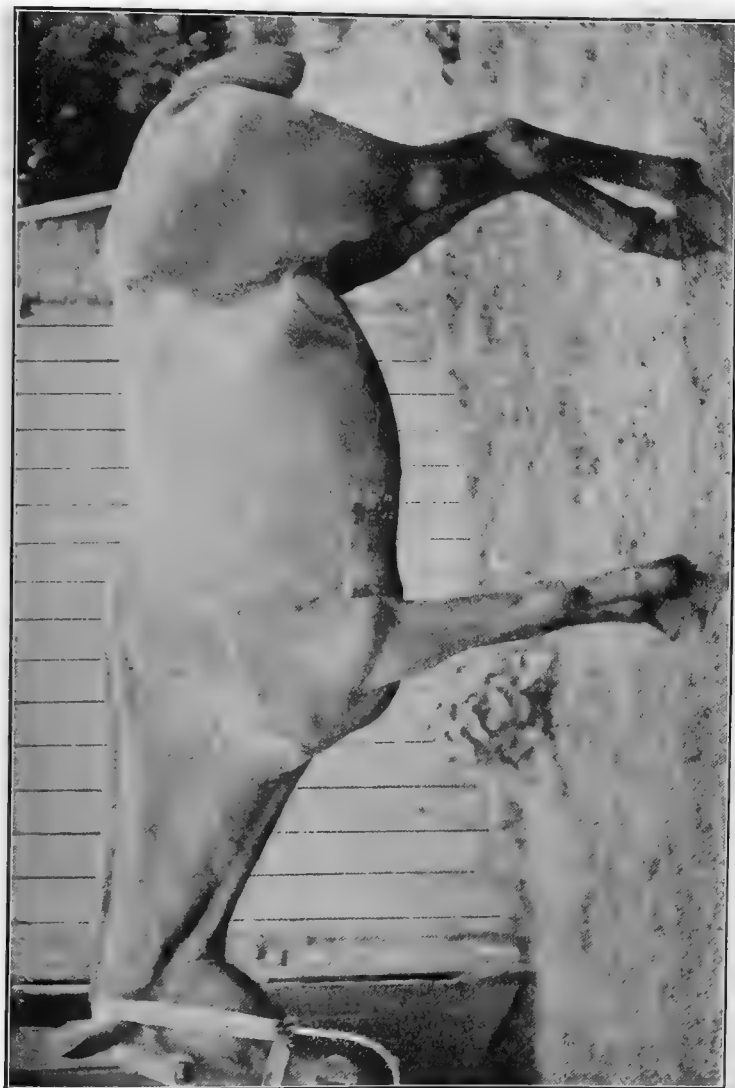


FIG. 22.—Epizootic or contagious lymphangitis.

## SYMPTOMS.

The onset of the disease is usually most insidious, and in this lies its greatest danger. It usually commences with what a stable-keeper would think was a girth or saddle gall, and its original mode of entry is usually at an abrasion occasioned by this means—*i.e.*, either by the saddle, collar, traces, bridle, or crupper. It is not until after it has been noticed that the wound shows no tendency to heal, or that the adjacent lymphatics are seen to be “corded,” that suspicion arises,

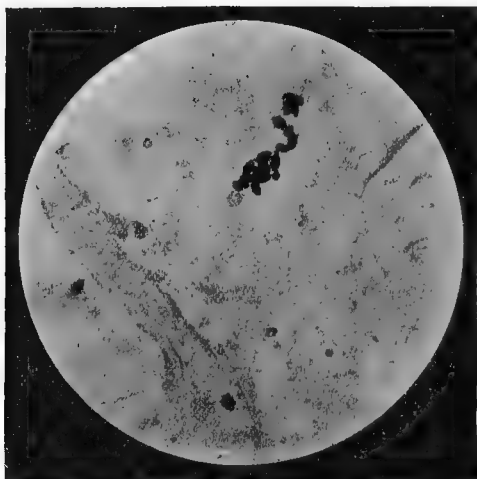


FIG. 23.—The *saccharomycosis farciminosus* of epizootic lymphangitis.

unless the disease is known to be prevalent at the time. The lack of a tendency to heal in a wound is always then suspicious; the subsequent cording of the lymphatics, and their nodulated appearance, will render it still more so. The symptoms then develop and the adjacent tissues become swollen; the nodes in the lymphatics get larger; in palpation they are soft, and appear to contain pus, and eventually burst. If a leg is affected, the limb will swell and show symptoms of subacute lymphangitis, with possibly enlarged glands. No time should be lost in making a microscopical examination, and, if possible, it is advisable to incise an unopened lymphatic bud and abstract some fresh pus from

near the walls of the abscess. The only conclusive positive symptom is the finding of the cryptococcus.

The general systemic disturbance is slight. In very acute cases there will be dejection, unthriftiness, remittent fever, and its concomitant symptoms, and somewhat rapid falling off in condition. In chronic cases there may be very little evidence except the actual lymphangitis.

Even in acute cases death may not occur for weeks or even months, and in the chronic form it may go on for years. The disease is not necessarily fatal, but the chances of complete recovery are remote. Many cases in which the lesions have healed and the animal apparently recovered have been known to break out afresh after some months.

#### TREATMENT.

If a case be treated in the very early stages by absolute extirpation of all diseased areas, there are hopes of preventing any recurrence; but even then it is doubtful, and it is now considered advisable to stamp out by slaughter and by thorough disinfection of all harness, litter, &c. If treatment be attempted, rigid isolation must be adopted and insisted upon for at least a year after all symptoms have disappeared. The incubation period is extremely uncertain, and it may not evince itself for many months. Its extremely contagious nature and insidious advance would then suggest stamping out as the most efficacious and cheapest method of treatment.

The disease is now scheduled by the Board of Agriculture (*vide* Regulations).

## CHAPTER XXV.

### CONTAGIOUS DISEASES—*continued.*

#### CANINE DISTEMPER.

*Definition.*—A febrile disease, due to the operation of a morbid poison, as the result of contagion and infection, and known in almost all parts of the world. The specific microbe has not been discovered, but it is known that the disease is transmissible by contagion and infection, and that, therefore, the virus is both volatile and fixed. It is most readily conveyed by the air. The virulence of the contagium contained in the discharges, the blood, &c., is diminished if kept in a dry state for a time, but congelation and desiccation seem to exert no power over it.

It is therefore one of those diseases which now make a considerable list—*i.e.*, one of those due to an ultra-visible organism.

*Cause.*—It is by many observers now conceded that distemper is due to an organism of the *Pasteurella* type, and this may be accepted. The work of Phisalix and Lignière have demonstrated this organism as the cause, and have made both a curative serum and a protective vaccine which have given good results. It has already been pointed out in connection with contagious pneumonia of the horse that the peculiarity of *Pasteurella*'s generally is that in the later stages of the diseases which they induce their presence may not be demonstrated either by microscopical examination or cultural tests. It does not follow, however, that they were not the initial cause; but having practically finished their work, they die off, and become replaced by other saprophytic organisms. This may be the case in distemper, as we know that in the later stages bipolar organisms in the lesions are as a rule in

the minority. The effects of the vaccines of Phisalix and Lignière, however, tend to show that the specific cause is undoubtedly a *Pasteurella*. These serums and vaccines must, to be efficacious, have a polyvalency; that is, they must be made from races of the organism obtained from a variety of sources, as an organism that is pathogenic in one locality may not be so in another, and *vice versa*. To obtain a true polyvalency, each variety of organism must be cultivated separately, and the products not "pooled" until the vaccines are ready.

#### PATHOLOGY.

The virus primarily induces a febrile condition of the system, and specifically affects the mucous membranes of the nose and eyes. In some cases the poison destroys by *shock*, the animal dying, in a few hours, from collapse, without any true signs of distemper being developed. In other cases the action of the poison is concentrated upon the nervous system, causing epileptic fits, spasms of various muscles, and finally coma and death. Again, the liver seems to be the organ chiefly affected, and in other instances the intestinal canal or the bronchial mucous membrane is the seat of the local effects of the virus. Secondly, the spinal cord or some of the spinal nerves become diseased, and, consequently, distemper often terminates in paralysis of the posterior extremities, or in chorea; the clonic spasm characteristic of chorea being generally more observable in the muscles of the neck and fore extremities. In consequence of the variety of forms which the malady assumes, it has been described as five different diseases, namely—(1.) The catarrhal; (2.) The respiratory or bronchial; (3.) The bilious; (4.) The intestinal; and (5.) The nervous. These are merely varieties of one and the same disease, and exemplify in a most remarkable manner the method in which the same virus or poison may act upon more parts or organs than one, as is the case with various medicinal and other agents.

The spasmodic condition of the body—the chorea—and the paralysis also illustrate how a morbid poison, after exhausting its powers upon one or more organs, may, after a space of time, affect other organs of the same body.

Distemper, like all contagious and infectious diseases, has an uncertain but short period of latency. It, as a rule, affects the system only once; hence it is most prevalent in young dogs, and sometimes prevails as an epizootic.

Some writers have compared distemper to the typhoid fever of man. I have carefully dissected numerous fatal cases of distemper, and looked particularly for the specific lesion of typhoid, namely, congestion and tumefaction of Peyer's glands, but have failed to detect any growth in the gland cells of the intestines. In the intestinal form of the disease a generally congested condition, with even ulceration of the mucous membrane, may be witnessed, but these ulcerations are very different to those observed in typhoid fever. Again, intestinal lesions are the specific distinctions of typhoid, whereas in distemper inflammation of the mucous membrane of the bowels is only seen in that form in which the virus seems to exert its influence on that part of the animal body, and it is only in a minority of instances that this effect is witnessed.

I can compare distemper to no human disease except measles, and the points of analogy are very great. In both diseases catarrhal symptoms are manifested; they are infectious diseases; they generally occur but once in a lifetime; they chiefly affect the young; in almost all cases of distemper there is some cutaneous eruption or rash, and desquamation of the cuticle; catarrhal ophthalmia, bronchial and pulmonary inflammation, and dysentery, are complications of both diseases, and, finally, convulsions sometimes occur both at the commencement and during the progress of measles and of distemper.

I am not aware, however, that measles is succeeded by either paralysis or chorea; nor do I mean it to be understood that distemper is communicable to man.

#### SYMPTOMS.

The primary symptoms are those of fever, associated with those of catarrh. The dog shivers, is dull, restless, with dry nose and injected eye. The appetite is partially lost; there is thirst and rapid loss of flesh and condition; the urine is high coloured and scanty; the bowels are generally irregular, sometimes constipated, sometimes looser than natural; the fæces are dark

coloured and foetid. In the course of a few days the catarrhal symptoms, which at first may have been limited to frequent sneezing, with a slight discharge from the nose and eyes, are fully confirmed. The nasal discharge is now more or less profuse; the eyes are weak, occasionally inflamed, and discharge tears and mucus. Very often the eyelids will be gummed together, and the animal thus rendered temporarily blind. Cough is present, at first dry and husky, afterwards moist. The breathing is sometimes much quickened, and the cough dry and painful, showing that the lung tissue and pleura are affected: the pulse may range from 120 to 150 beats per minute, and the temperature is elevated. In other cases the respiratory movements are but little affected, except when the bronchial tubes are filled with mucus, which is coughed up, and the breathing becomes much relieved. As the disease advances, debility rapidly increases, the dog being often at the end of the first week scarcely able to stand; the appetite becomes more and more impaired, and the digestive powers much debilitated. Food now partaken of or forced upon the animal is quickly ejected by vomiting, or passes through the intestinal canal in a foetid, ill-digested condition. At the end of about a fortnight these symptoms may abate in intensity, and the dog slowly regain its strength. Very commonly, however, complications occur which tend towards a fatal termination. The complications are as follows:—

1st. *Pneumonia*.—The breathing becomes laboured, rapid, and panting; the prostration of strength is very great; the dog is unconscious of surrounding objects; the pulse is frequent, feeble, and intermitting, and the feet, nose, and ears are icy cold. If the ear be applied to the chest the crepitating sounds of pneumonia will be detected.

2d. *Jaundice*.—This occurs from the presence of a blood poison, interfering with the normal metamorphosis of bile, from congestion of the liver, or most commonly from catarrhal inflammation of the mucous membrane of the biliary ducts; the swollen mucous membrane mechanically blocking up the tubes, and thus impeding the flow of bile.—(See *Liver Diseases*.)

3d. *Intestinal complications*.—Vomiting and purging are prominent symptoms. Sometimes there is true dysentery, the faeces being tinged with blood; tympanites and abdominal pain.

*4th. Epileptic fits.*—Spasmodic convulsions of varying intensity, coma and paralysis, more or less complete, occur as primary symptoms. These are to be separated from those signs of nervous alterations which occur as secondary affections in distemper. In the first case the brain and nervous symptoms are concomitant with or shortly succeed the attack; whilst in the latter, chorea, paralysis, or complete coma appear after the febrile condition has more or less abated, and when the dog seems in a fair way to recover.

*Conjunctivitis* is not at all an uncommon complication, and ulceration of the cornea, perhaps unpreceded by any inflammatory signs, may occur from mal-nutrition.

In other cases cellulitis or an erysipelatous inflammation of the extremities occurs, the inflammation sometimes suppurating in various parts: the suppuration, being of a diffuse or infiltrating character, causes much pain and rapid exhaustion.

In most instances some cutaneous eruptions are seen on the inner surfaces of the thighs and other parts, where the hair is thin and downy; the eruptions at first resemble flea-bites, but may become vesicular or even pustular; the skin is harsh, and much epithelium is thrown off, causing the hair to be filled with scales of scurf.

There are various conditions of the body which predispose the disease to attack the dog in the various ways above enumerated. The presence of worms in the intestinal canal may excite the intestinal form; previous high feeding and obesity the bilious; and the eccentric irritation of the nervous system caused by teething, worms, &c. predispose the dog to the nervous form.

Distemper originates undoubtedly from contagion as well as from atmospheric infection, and usually rages as an epizootic. The majority of dogs in some districts suffer from the disease, whilst in other parts of the country it is scarcely ever heard of, unless brought there by a dog already diseased, and no class of dog is exempt. During the author's residence in Australia the disease was imported from Europe, and scarcely a dog in the colony of Victoria was unattacked. Nor were the wild dogs allowed to escape; hundreds of these were to be seen lying dead in the bush in various parts. It is also stated that it attacks the cat, wolf, hyæna, prairie dog, and monkey.

The contagium or virus, probably a microbe which may yet be



attenuated and applied for the purpose of protective inoculation, will taint a kennel for a long time after the disease has disappeared; and it is always unsafe to introduce fresh dogs into such a kennel, if it cannot be proved they have passed through the disease, unless the walls, drains, fittings, &c. are thoroughly cleansed and disinfected.

#### TREATMENT.

In the treatment of distemper it must always be remembered that the disease runs a certain but indefinite course, and that all the symptoms are but the result of the operation of a morbid material existing in the blood. If these facts be borne in mind, the practitioner is not likely to resort to those dangerous and exhausting remedies so generally recommended by writers on canine diseases. The administration of calomel, jalap, aloes, tartar emetic, digitalis, with the application of blisters or setons, is calculated at all times to do harm.

In the earlier stages, if the bowels are at all irregular, a small dose of castor oil is to be prescribed. The dose must vary in strength according to the size and age of the dog; from a tea-spoonful for a small dog or young puppy, to an ounce for a well-grown dog. This will remove any ill-digested or indigestible material from the intestinal canal.

After the laxative has operated, I have found from two to six grain doses of the hyposulphite of soda useful, modifying the symptoms most materially, and converting what has threatened to be a serious case into a mild attack. Care must always be taken not to administer any medicine in such large doses as to disorder the stomach in any way, or to cause vomiting, as it is of essential importance to keep up the animal strength by proper food, *spontaneously partaken of*. If there be much prostration of strength, a tea-spoonful of good sherry, or one drachm of spirits of nitrous ether, may be administered three or four times a day, in addition to the hyposulphite, with very great advantage.

This simple treatment, in addition to warmth, fresh air, a clean bed, and clothing for thin-haired dogs, with a plentiful supply of fresh cold water (or milk and water if the dog will take it) for the animal to drink, sponging of the nose and eyes not being forgotten, will often be successful. The food must be restricted in quantity, and of an easily digested nature. In

my own practice I find porridge and milk to be the best, provided the dog will take it; but if it be a pet dog, and used to pampering, it must have what food it will eat, and what it has been used to, in modified quantities. It may be laid down as a rule that the digestion of what the dog is fondest of will be more easily performed, provided that it is not allowed to eat too much. After the disease has continued for six or seven days, small doses—from one grain to three—of quinine may be administered, care being taken to discontinue it if it seem to disagree with the dog.

If there be much irritation of the stomach and vomiting, hydrocyanic acid, from two to four drops, Scheele's strength, will have a calmative effect on the gastric organs and allay the vomiting. Purging, if not excessive, should not be violently checked, but should the dog seem to lose strength from this cause, chalk may be first tried, and afterwards tincture of opium, from ten to twenty drops, three times a day.

The pulmonary complications are best relieved by the application of hot flannels to the sides, or hot fomentations may be substituted in smooth-haired dogs. In rough-haired ones, the hair saturated with the wet is a long time in drying, and the animal is apt to take cold. Nitrate of potash may be dissolved in the dog's drinking water, or given in solution in from six to twenty grain doses, as a febrifuge and diuretic; and the chloral hydrate may be given at night, particularly if the dog is sleepless. The nervous symptoms may depend upon reflex irritation. If from teething, the gums are to be lanced. If from the presence of worms, and this is a common cause, and tape-worm the parasite generally met with, from one scruple to one drachm of areca-nut is to be administered. If the appetite is entirely lost, advantage must be taken of the thirst, and beef tea or milk allowed the dog to drink. If it will not drink spontaneously, nourishment must be forced upon it, in small quantities and oft repeated. A raw egg beaten up with a glass of sherry, carefully administered, will be of great service; in other cases, brandy and beef tea. I am, however, opposed, unless the prostration of strength threatens to prove rapidly fatal, to the forcing of large doses of stimulants, as they often cause much mischief, careful nursing and good attendance being much more beneficial than any mere medicinal remedy.

I have very little to say upon the treatment of the secondary nervous complications. They are due to a variety of pathological changes.

Paralysis sometimes results from obliteration or plugging of the spinal veins, which are found enlarged and pressing upon the cord: I have found this in several dissections of dogs which had died or had been destroyed whilst suffering from paralysis—from atrophy of the cord, the nervous matter of which being in some instances replaced by a semi-fluid deposit, and from congestion and disintegration of its substance.

Some cases of paralysis after distemper, if kept long enough and well fed, will recover. As a rule, however, the loss of power remains, and the dog has ultimately to be destroyed. I have tried strychnia and other nervine tonics, but cannot say that they do much good. Iodide of potassium—given on the assumption that the paralysis resulted from the pressure of an exudate on the cord—has been tried also by me, but with no very successful results. Blisters to the spine, setons, and even the application of the actual cautery, have also been tried in such cases. Such treatment inflicts much pain on the poor patient, but does not remove the disease or prolong life.

If the eyes are tender or inflamed, they are to be bathed with a solution of boracic acid; and, in order to relieve any pruritus and prevent scratching, a five per cent. solution of cocaine should be applied every few hours. Irritants, such as the nitrate of silver and sulphate of copper, should be avoided. Calomel, blown into the eye, may be tried in chronic opacity.

Within the past few years it was thought that immunity might be conferred in dogs by the injection of a series of virus vaccines of various degrees of virulence, and the method is known as Phisalix's vaccination. Unfortunately, however, McFadyean, Gray, and others have found that it is useless as regards dogs in Great Britain. (For full details of this form of vaccination, see "Blood-serum Therapy," by Jowett; Baillière, Tindall and Cox, London.)

## CHAPTER XXVI.

### CONTAGIOUS DISEASES—*continued.*

#### ANTHRAX.

ANTHRAX; charbon; gloss-anthrax; apoplexia splenetica; carbunculo contagiosa, &c. (L.); charbon; chancre à la langue; mal de sang; sang de rate; typhomiè; fièvre putride, &c. (F.); miltzbrand; miltzbrand-fieber; petechial typhus; pestfieber (G.); carbone; febbre carbonculara, &c. (I.); apoplexy of the spleen; malignant sore throat; known in India as Loodiana disease, and in South Africa as horse sickness; in sheep as splenic apoplexy; in America as splenic fever, Texan fever, trembles, &c.

The term *charbon* is applied by the French veterinarians for the reason that the regions of the body where the disease is localized are coloured black. Anthrax (a burning coal) is now adopted by most writers as a generic term, and applied to what is otherwise known as splenic fever; but it throws no light on the nature of the disease, as others, septic and putrefactive in their nature, present a similar appearance of the blood.

*Definition.*—The disease is a true septicæmia, and consists in a special and primitive alteration in the blood, in which an organism termed the *Bacillus anthracis* is rapidly developed and propagated, and is more special to the herbivora and birds. Inoculation with the blood or tissue of animals which have died from it induces some one or other form of the disease,—in man, as a rule, malignant pustule. For this reason anthrax is looked upon and described as a truly contagious disease.

Anthrax appears at all seasons, but principally in the spring or during summer and autumn. It occurs either as a sporadic, enzootic, or epizootic disease, attacking animals of any age—the fat, vigorous, plethoric, as well as the lean, feeble, and languid,

It is a remarkable fact that wounds, simple in themselves, in cattle subjected to the influence causing charbon, although not suffering from it, often become mortal.

#### HISTORY.

Anthrax has a very ancient history, and was known in Asia Minor at the period of the siege of Troy; but, leaving ancient history aside, it may be useful to mention that the seventeenth and eighteenth centuries were remarkable for the devastations committed by various epizootic outbreaks of anthrax. In 1617 it was prevalent and of such a fatal nature in the neighbourhood of Naples, that over 60,000 persons perished through partaking of the flesh of animals which had died of the disease. In 1731 it declared itself in several provinces of France, notably in Auvergne, Bourbonnais, and in Languedoc, where it was studied by Sauvages, and described by him under the term *glossanthrax*.—(*Nosologia Methodica*, vol. ii. page 360.)

1757, 1763, 1775, 1779, 1780, and 1800.—These years were signalized by a charbonous malady which extended nearly all over France, and affected all the domesticated animals. The disease was studied by Bourgelat, Chabert, Berdin, Huyard, Desplas, Detil, Gordine, Gilbert, and a great number of veterinarians. From 1800 to 1846 many outbreaks of charbonous disease were observed, generally in the hottest months. They were studied by Demoussy, Sansol, Pradal, D'Arboval, Mathieu, and others. During more recent periods, outbreaks have been studied by Roche-Lubin in Avignon; by M. Rey on the heights of the Alps; and in Eure et Loire by the Medical Society of the department; and by MM. Renault and Delafond, who were sent by the French Government, the one to Allier and Nièvre, and the other to Somme, to report on the disease. Within recent years the disease has been profoundly studied by Pasteur, Chauveau, and Koch, and others in France and Germany. In this country, however, little has been done for its elucidation; and though less frequent here than on the Continent, it is, however, quite as fatal in its character.

#### ETIOLOGY.

The influences which predispose to the development of anthrax are arranged by French veterinary writers under four

principal heads, comprising respectively the influences of temperature; water spread over the surface of the earth, as in morasses and stagnant ditches; forage tainted with decomposing animal and vegetable matters; and contagion.

*Influence of temperature.*—The hygrometric and thermometric conditions of the atmosphere, which always exercise a marked action on the organism, disposing it under certain circumstances to anthrax, are stated by several authors to be those characterised by humid and persistent fogs, coldness, and humidity; a tempestuous atmosphere; alternations of burning heats and stormy rains.

It rages as an enzootic on the borders of rivers, and in low lands which have been inundated. In the months of July and August, signalized by excessive heat, charbon has been frequent. The years 1712, 1731, 1775, 1779, 1780, 1823, 1824, 1825, 1846, have furnished memorable examples. Under the influence of excessive and prolonged heat, the rivers, ponds, brooks, &c. were dried up, the soil opened in crevasses, and the disease was developed to an enormous extent. In France it is stated that a very warm summer is never seen without charbonous diseases being prevalent; and it is concluded that a high temperature, especially if preceded by a damp or moist atmosphere, is very favourable to the development of charbon.

*Influence of ponds, morasses, and stagnant waters.*—The history of the malady demonstrates that morasses are favourable to its development, as it is observed that it is frequent in countries where they occupy a large surface. It is also frequent in countries exposed to inundations, and where water stagnates on the surface of the soil; and where animals are made to stand in mosses and stagnant waters, the malady commits great ravages. This fact is remarkable, not only in France, but in other countries, particularly in the Mississippi valley in America, and in Africa and India.

Observers who have closely watched these affections in this country, where it seldom appears in the horse, almost unanimously conclude that in cattle and sheep they are due to dietetic errors; more particularly to sudden and violent changes in diet, whether that change be from a poor to a highly nutritious, more particularly a nitrogenous diet; from dry and good food to watery unripe provender; to damaged food of any kind; the

influence of undrained lands; defective ventilation and drainage of stables; to food and water contaminated with the morbid products of animals which have died of anthrax. In one remarkable outbreak which came immediately under my notice, the disease appeared amongst sucking calves of the pure short-horn breed, and which had never partaken of other food than what they obtained by sucking, the dams remaining healthy. Anthrax is also disseminated through the agency of flies; and Bollinger, who has observed that the disease is often most prevalent when flies are in the greatest abundance, has induced it in rabbits by inoculating them with flies caught on the carcasses of animals dead from anthrax. The flies, however, resist the influence of the virus, although bacteria are found in them. Pasteur's assertion that the spores of anthrax are brought to the surface of the ground by earth-worms—contested by Koch—is now confirmed by Bollinger, who has found that five per cent. of the worms coming from an infected pasture-ground contained the spores of anthrax.

The theory as to spores being conveyed by earth-worms is open to serious doubt. The bacillus of anthrax is strictly *aërobic*, and will only sporulate at a temperature of about 30° C. (86° F.). If an animal affected with anthrax is buried without opening the carcase, all the organisms are exposed to *anaërobic* conditions, and therefore cannot grow or sporulate. Previous to death no sporulation will take place in the body. It would seem, then, that no spores are buried, and as the temperature of the soil is never as high as 98·6° F., in England at any rate, the possibility of such means of contagion is very remote. Contagion must therefore be by the careless disposition of diseased carcasses, thus blood or contaminated material is exposed to *aërobic* conditions, sporulates, and so infects the soil. These spores remain alive indefinitely under ordinary conditions until an opportunity is afforded them of infecting an animal. The earth-worm theory is a possibility, but rather an improbable one. It will be seen, therefore, that if an infected dead animal is buried without the skin having been broken, or without in any way giving the infected tissues *aërobic* conditions, then all danger is exempted, and neither lime nor other disinfectant is necessary. As a safeguard, however, disinfectants or fire are invariably used.

Dogs, cats, white mice, and Algerian sheep are said to have an immunity from the disease.—(CROOKSHANK.)

In opposition to the view of the spontaneous origin of anthrax, we have the observations of many eminent pathologists—and this view is now universally accepted—who maintain that the malady is due to the propagation of a now well-known aërobic organism, the *Bacillus anthracis*, the history of which is as follows:

These organisms were first observed by Brauell, and afterwards by Delafond and Gruby, in the blood of animals which had died of anthrax, as peculiar staff-shaped bodies, which Delafond designated *bâtonnets*, and which were believed to be products of putrefaction, and that anthrax was a septicæmia or putrefaction of the blood. These *bâtonnets* were afterwards observed in 1850 by MM. Davaine and Rayer, and some time later Koch studied them, and found the aqueous humour of the ox's eye to be particularly suitable for their nutrition. With a drop of the

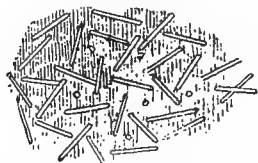


FIG. 24.—Transparent rods.

aqueous humour he mixed the smallest speck of a liquid containing the rods, placed it under the microscope, warmed it suitably, and watched the subsequent action. During the first two hours hardly any change was noticeable, but at the end of that period the rods began to lengthen, and the action was so rapid that at the end of three or four hours they had attained from twenty to thirty times their original length, and at the end of a few additional hours had formed filaments in many cases a hundred times the length of the original rods; and further, it was seen that within the transparent rods little dots appeared; these became more and more distinct until the whole organism was studded with minute ovoid bodies like peas within their shell. After a time the integument fell to pieces, the place of each rod being taken by a long row of seeds or spores. Koch concluded that these spores, as distinguished from the rods, constituted the contagium of the disease in its most deadly and persistent form.

By inoculating animals with the fresh blood of an animal suffering from splenic fever, he found that they invariably died within twenty to thirty hours after inoculation. By drying the infectious blood containing the rod-like organisms,



in which, however, the spores were not developed, he found the contagion to be fugitive, maintaining its power of infection for five weeks at the furthest. He then dried the blood containing the fully developed spores, and exposed it to a variety of conditions. He permitted the dried blood to assume the form of dust, wetted this dust, allowed it to dry again, placed it for an indefinite period in the midst of putrefying matter, and subjected it to other tests. After keeping this spore-charged blood, which had been treated in this fashion for four years, he inoculated a number of mice with it, and found its action as fatal as that of blood fresh drawn from the veins of an animal suffering from splenic fever, each spore in the millions con-

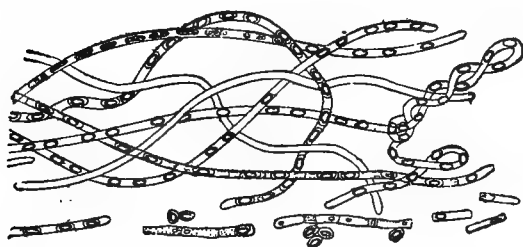


FIG. 25.—Spore-bearing filaments.



FIG. 26.—Spores.

tained in the diseased blood being sufficient to produce the disease.

The bacilli are not always found in the blood of living animals suffering from the disease; indeed, they generally appear a few hours before death, which never takes place in less than twenty hours, and then only singly and in very small numbers. Their number, however, varies with the animal inoculated; in the guinea-pig they are numerous, sometimes exceeding the blood corpuscles; in the rabbit much smaller, and in the mouse often absent altogether. If the disease has been induced by inoculation, they are present, though in variable numbers, in the inoculation carbuncle. Though the rods are not always found in the blood, the spores are said to be invariably present, and some assert that it is their product that destroys life.

The bacilli rods are straight or somewhat irregularly outlined, measuring from  $\frac{1}{25000}$  to  $\frac{1}{35000}$  of an inch in breadth, but they

vary in length very considerably, those in the spleen being longer; the shortest rods being in length generally about twice the diameter of a human red corpuscle, the longer ones two or three times the length of the shorter\*; but when carefully examined, the latter will be seen in a process of division into two or more segments. They are broad at each end, truncated, and slightly concave, and when united the concave ends enclose a lenticular space; they are non-motile, and their ends are not rounded, but terminate sharply.

The bacillus of anthrax is easily stained by any of the aniline stains. The stain preferred, however, is old solution

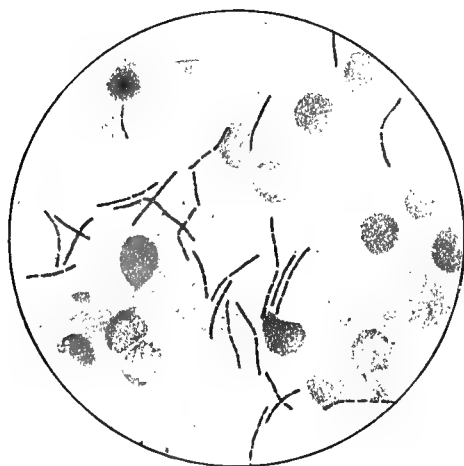


FIG 27.—Anthrax bacillus and blood corpuscles.

of ethyl blue, because with it there is a peculiar reaction which is not noticed when other organisms are stained with this dye.

McFadyean and a German pathologist simultaneously observed that when using this stain in anthrax blood-smears the stain split up, the bacilli were stained blue, and the fluid around them stained pink. In carefully prepared specimens this is so marked that the smear seems stained more pink than blue if held up to the light.

Another important fact to be remembered is that anthrax bacilli have well-marked capsules which are unstainable, and

in a typical specimen we should see with methylene aniline blue a blue clean-cut rod surrounded by a clear space, and this again surrounded by pink.

According to the observations of Koch, it appears that, whatever be the species of animal inoculated with anthrax blood, and no matter how many successive inoculations may be made, the *bacilli* multiply solely by fission, but only so long as the animal is alive; when dead a minute portion of its blood, placed in aqueous humor, and kept at a temperature of 35° to 37° C. (95° to 98½° F.), the rods, as already stated, lengthen out very considerably. This process of lengthening of the rods into filaments is apparently effected by the temperature. In five hours a rod at a temperature of 32° C. (89·6° F.) may have increased so as to be from eighty to one hundred times its original length, and in twenty-four hours the filament may be full of spores. If the temperature, however, be kept about 28° C. (82·5° F.), the spores may not appear till the thirty-sixth or fortieth hour. When the spores have once appeared, all the other changes go on at ordinary temperatures from 12° C. (53·6° F.) to 18° C. (64·4° F.), but not nearly so rapidly, even when the preparation is kept in the sun for a few hours daily, as when artificial heat is applied. On the other hand, a high temperature, 37° to 40° C. (98·6° to 104° F.), at once checks all developmental changes.

The filaments differ in cultivated specimens very much in their arrangements. Sometimes they form a network—indeed a mycelium—made up of numerous, nearly parallel, unbranched threads, crossing each other at different levels; the threads are sometimes straight, but have generally a wavy outline. This condition may obtain throughout the whole preparation, but generally at some parts the filaments are extremely irregular and much convoluted.

Pasteur stated that the spores of bacilli remained toxic after boiling; and after being subjected to a pressure of twelve atmospheres of oxygen, Dr. Burdon Sanderson and Dr. Cossar Ewart tested the accuracy of this statement, and found that mice inoculated with the boiled and compressed solutions remained quite well.

The experiments of Bert, however, support to some extent

the conclusions of Pasteur. In a series of experiments Bert submitted anthrax blood to the action of considerably compressed oxygen, and found the bacilli had disappeared, killed by the oxygen, and yet the blood retained its virulence, for it killed rabbits, guinea-pigs, and dogs inoculated with it; and in another series of experiments Bert took anthrax blood containing bacilli, and added drop by drop of absolute alcohol to it, until a precipitate was formed, and which was dried in vacuum. This dried powder was injected under the skin, and it killed a rabbit, a guinea-pig, and even a dog.

If this alcoholized precipitate be dissolved in water and filtered, the filtrate is still virulent. If alcohol is again added to it, it forms a flocculent matter, which is deposited at the bottom of the vessel. Collected on a filter and dried, this precipitate is still toxic. It would therefore appear that the active or virulent element of anthrax resists absolute alcohol, and that it resists oxygen, and that it is precipitated by alcohol and soluble in water. It behaves itself something like diastase, except that, whatever may be its nature, it can reproduce itself to an indefinite extent; while it is asserted by some authorities that diastase cannot reproduce itself.

Putting aside the conclusions of Colin—that the bacilli are simple transformation of the blood-corpuscles—we are left between two sets of conclusions. Those of Koch and others point to splenic fever being due to a minute organism possessing wonderful powers of resistance and reproduction; Bert's to something independent of the presence or absence of animal or vegetable organisms—a ptomaine—which resist the action of compressed oxygen and absolute alcohol, and which would, he asserts, kill everything possessing life: this something he is of opinion is a substance analogous to diastase.

Blood containing bacilli, if dried in very thin layers, by being exposed to the air in a shady place, was found by Koch to lose its virulence and its power of developing elongated fibres after twelve to thirty hours. Thicker layers retained their powers for two or three weeks; and some still thicker for four or five weeks. After a longer time they were never capable of producing the disease.

Koch also found that if the bacilli were deprived of air they soon died.

When rubbed up with the blood or aqueous or vitreous humor of an ox, and placed in a well-closed glass vessel, there quickly ensued an odour of putrefaction; the bacilli disappeared after twenty-four hours without the fibres enlarging, and lost their infective power. That their death was due to the absence of oxygen was shown by placing a drop of blood infected by the bacilli under the microscope. Examined by the micro-spectroscope it gave the bands of oxy-hæmoglobulin; the fibres in this drop increased four or five times in length in three hours, but after that time the oxygen was clearly used up, as the presence of the absorption band of reduced hæmoglobulin proved. From this moment the growth of the bacillar fibres ceased, although true putrefaction had not set in.

When the spores and bacilli are separated from the blood by filtration, the blood is said to be rendered innocuous; and when pregnant animals become affected, or have been inoculated, the blood of the foetus does not become diseased, and other creatures can be inoculated with it and suffer no harm, the intervening membranes acting the part of a filter. The bacilli also disappear in liquids in the presence of carbonic acid, and the blood soon loses its specific property.

This proves that, to live and grow, the bacilli require to absorb oxygen and give out carbonic acid; hence they are what M. Pasteur terms "aërobic." If the fluid which contains them begins to putrefy they are destroyed, not only by being deprived of oxygen, but by being brought into contact with other organisms, such as the microbes of putrefaction, in the presence of which, and of all other low forms of organisms, they either do not develop at all, or develop with great difficulty. The organisms of putrefaction are not aërobic, and cease to move when brought in contact with oxygen; disappear, being transformed into refracting corpuscles, which in a suitable soil become motile, and multiply with extreme rapidity in a putrefying fluid. If an animal be inoculated with it when in this condition, it does not die of anthrax, but of septicæmia, the symptoms of which, when produced in guinea-pigs with the blood of a horse which had been dead of charbon twenty-four hours, and which contained the bacteria of putrefaction as well as some bacilli, and with the blood of a cow which had been dead forty-eight hours, and which contained a preponderating quantity of motile

organisms, were violent inflammation of all the muscles of the abdomen and limbs, and here and there, especially on the ears, bullæ formed containing gas. The blood was diffuent, and on examining these animals immediately after death, M. Pasteur found that the muscles were filled with active vibrios of putrefaction, and in the peritoneal cavity they had undergone extraordinary development; one drop of this serosity taken from an animal still living affected another animal profoundly, while a drop of blood from the heart had no effect. The spores are very tenacious of life, resisting many germicides. The rods are destroyed by dry heat at a temperature of 212° F., whilst the spores require to be exposed for three hours to a dry temperature of 283° F. before they are killed. Some writers assert that the spores resist the action of boiling water, but Hamilton says that they are killed after a few minutes' boiling: the rods, however, perish in a moist atmosphere at a temperature of about 140° F.

Putrefaction and the action of carbonic acid gas, whilst destructive to the non-sporulated rods, have no effect upon the spores themselves; both spores and rods are, however, killed in ten minutes by corrosive sublimate—1 per 1000 solution—within twenty-four hours by a two per cent. solution of chlorine, bromine or iodine, and strong sunlight; but they seem to resist five per cent. solution after twenty days' exposure, twelve days phenic acid five per cent., and nineteen days ten per cent. solution of lysol. Iodoform seems to have no influence either on the spores or rods. Whilst the rods are destroyed by putrefaction, it is otherwise with the spores, which have a much greater degree of resistance, and it is due to this retention of virulence by the spores that anthrax continues in buildings, grass lands, dried fodder, and water. Grass and hay grown upon land where anthrax carcases have been buried months before have conveyed the disease, and the same may be stated of water, particularly spring water obtained from wells situated at a considerable distance, but below the graves of such animals.

Again, dilution of the fluid containing the bacilli with a moderate amount of water has no effect on its virulence, but a large quantity destroys it, and traces of carbolic acid prevent the development of the bacilli.

It has been stated that the bacilli destroy life—(1.) By acting

as asphyxiants, depriving the blood of its oxygen ; and (2.) By mechanically obstructing the blood-vessels. Against both these theories must be placed the fact that they are very few in number, indeed often absent altogether in the blood during life.

Anthrax is not transmitted by infection from one animal to another, for animals kept in the closest proximity to diseased ones, and placed under the most favourable conditions for infection through the air, do not become diseased.

Mice and rabbits seem capable of eating food containing bacilli with impunity, and flies can gorge themselves with the infected blood and suffer no harm ; but horses, cattle, pigs, dogs, cats, guinea-pigs and ferrets succumb after partaking of food and water contaminated with the virus.

It is now generally admitted that animals are infected by spores contained in the food, which gain entrance into the circulation either through abrasions in the mucous membranes of the digestive track, or, as some assert, by the pulmonary mucous membrane ; but this is doubtful. My own experience leads me to conclude that uncorticated cotton cake is the most fertile source of the disease of all the artificial foods, and it seems to acquire virulence after being kept for some time, particularly if neglected and allowed to become heated and mouldy. When freshly made, it seems to have no effect, and I have known several instances where cattle have eaten the same cake for weeks or even months before becoming affected, then all at once they have died off. I am of opinion that the spores—few in number—have been present in the cake from the first, and that they multiplied in the cake, which, as is well known, is imperfectly dried ; thus, after repeated reproductions of the organisms, the cake has become sufficiently charged to induce the disease in its partakers. The decorticated cotton cake is differently prepared, being highly dried and submitted to great pressure ; thus heating and fermentation are prevented, and any increase of the organisms rendered impossible.

When soil is contaminated with the blood or discharges of animals dead of anthrax, it is found that spore formation goes on actively in many moist and warm media, and it is stated that these spores may be conveyed by floods to surrounding pastures. It is also supposed that the organism may grow as a saprophyte

on dead vegetable matter. Whether this be true or not, the vitality of the organism must be very tenacious, as it is well known that certain pastures are always dangerous to cattle, but more particularly after rain preceded by warm dry weather.

It is most remarkable that preventive inoculation has not succeeded in this country, although so successful on the Continent. Is it the fault of the operators, or what? A reference to experiments of Dr. Burdon Sanderson and Mr. Duguid (see below) ought to convince any one that the operation should be further tested, and that the Government should encourage all experiments calculated to increase the general welfare, instead of throwing obstacles in the way.

The local effects of inoculation of the skin with anthrax blood is as follows:—In twenty-four hours there is redness of the spot, with heat, swelling of the skin and subcutaneous tissue, extending from a third of an inch to an inch in depth.

The swelling increases in forty-eight hours to perhaps two inches, and on the third day, if the animal survive, to several inches; the heat and redness being most intense at the inoculated spot. The process extends in the connective tissue, particularly along the track of the lymphatics. In superficial inoculations bacilli can be seen in every instance in twenty-four hours, at a distance only of about one-fourth of an inch, but their after extension is not proportionate to the extent of the tumefaction, nor does the serum found in the swelling contain many of them until after forty-eight hours, when great quantities will be found in it; when the virus is injected into the subcutaneous tissue death may occur without bacilli being found at the point of injection.

#### INOCULATION FOR THE PREVENTION OF ANTHRAX.

In 1878 it was discovered by Dr. Burdon Sanderson and Mr. Duguid that cattle might be inoculated with splenic fever from a guinea-pig, and though such inoculation caused the development of serious symptoms, the animals did not die; and in continuing these experiments it was found that cattle once so inoculated resisted the results of further inoculation,—that in fact they could be thus rendered insusceptible to future attacks of splenic fever. Dr. Greenfield, in making a series of experiments with the view



of obtaining a suitable virus for inoculation, found that the virus modified by transmission through the guinea-pig, and, cultivated under particular conditions, gradually lost its activity, and at last became practically inert; and it occurred to him that, by making use of this fact, a virus might be obtained so far modified as to be sufficient to ensure protection and yet not endanger the life of an animal inoculated with it, and this he found could be done with success.

The priority of this discovery is therefore claimed for England, but the merit of working out its details is undoubtedly due to Pasteur.

#### VARIETIES OF ANTHRAX.

The more recent investigators are disposed to confine the term anthrax to that form of disease characterised by black tumour, associated with the presence in the economy of the *Bacillus anthracis*; but as the term merely indicates a symptom—namely, a black tumour, the charbon (coal-like) of the French—I intend to include under it all those diseases in which it is an expression or a symptom, contenting myself by pointing out in which of them the bacilli have been discovered, and which are proved to be contagious.

Chabert designates anthrax or charbonous diseases as foul affections of different natures, and external tumours terminating in mortification of the tissues, and divides them into three kinds, namely:—

1st. *Charbonous or Anthrax Fever*.—In this form there are no external manifestations, and it is rapidly fatal; the *post mortem* appearances being engorgement and congestion of the spleen, liver, mesentery, intestinal mucous membrane, the sublumbar tissues, lungs, and heart. In some cases death is preceded by roughness of the coat, dry, hot skin, excessive sensibility over the dorso-lumbar region, choking cough, glairy discharge from the nostrils, and a quick, strong pulse. Sometimes the animal may live long enough for level tumours to form under the skin; but very often the patients succumb without presenting the least symptom of the malady, or in a few hours after the first appearance of illness.

#### ANTHRAX IN THE HORSE.

Anthrax in the horse rarely occurs in this country, but is

prevalent in India, where it attacks the elephant as well as other animals.

*Symptoms.*—The animal may appear dull, walking with a heavy, feeble step, then falls prostrate in a state of somnolence; if it be standing, the head hangs down, resting on the manger or other solid body. It sometimes stands back in the stall, resting the body on the side, and finally becomes restive, stamps with the feet, looks to the sides and flanks, and shows other signs of colic, and the temperature is much elevated.

If the disease comes on whilst the animal is at work, added to the above symptoms there is extreme lassitude, great weakness of the lumbar muscles and posterior extremities, with staggering gait. The skin has lost its suppleness, is hot, and slightly crepitates on the back, over the kidneys, and sides; the coat is rough and bristly in some parts; and there are partial or general tremblings of the muscles, and flow of saliva from the mouth. There are sweats, alternating from hot to cold. At the base of the ears, and behind the elbows, the veins become augmented in volume. Above all, the lymphatic ganglia of the groin are swollen; and if the horse be entire, the testicles move rapidly up and down. Great excitement now sets in, the animal is irritable and timorous, and afterwards becomes unconscious of all around. The conjunctivæ are yellow or reddish-yellow; and sometimes petechial spots are present on the visible mucous membranes.

The pulse is small and thready; the beatings of the heart are, however, strong, and are accompanied by a metallic tinkle. The respiration is often irregular, and often associated with roaring.

These symptoms may insensibly disappear, or may be succeeded by a critical eruption. At other times, even after the animal has seemed to rally, aggravation of the symptoms takes place. The animal grinds its teeth, has violent colic, rolls about, carrying its head to its flanks; the muscles of the head and neck are agitated by convulsive movements; the eyes are haggard and wandering; the mucous membrane injected, and of a brown or red tinge; the heart beats with extreme violence, very irregularly, and accompanied by a strong metallic sound; the pulse is trembling or double, and very small; the respirations tumultuous and agitated; the nostrils dilated, and clots of yellow (almost sulphur coloured) serosity and blood escape

by the nose; the mouth is filled with a mucous foam, the tongue is tumefied, and of a deep bluish-red colour (GLOSS-ANTHRAX); tears, sometimes tinged with blood, flow from the sunken and haggard eyes. The belly is sensitive when pressed upon, tympanitic, and the excremental matters are often liquid and mixed with blood clots; the rectum is often everted, appearing as a tumour, folded and livid; the temperature of the skin is lowered; the countenance is particularly anxious, the face shrunk. The muscular force now becomes exhausted; the animal falls to the ground; convulsions come on, more particularly of the neck and extremities; and finally it succumbs, after a few moments of calmness, which, succeeding a paroxysm, always precedes death. The disease may terminate in from six to forty-eight hours after the manifestation of the first symptoms. The ordinary time is from twelve to twenty-four hours, unless external eruption eliminates the morbid material from the body.

In the spring of the year 1884 a remarkable outbreak of charbonous fever, presenting the salient symptoms of Loodiana disease, occurred in a large stud of cart-horses under the care of Messrs. Leather, veterinary surgeons, Liverpool, and which had been, for some time previously, fed on an Indian pea (*Pisum sativum*), called in Liverpool Indian mutters. *Mutur* is the Hindustani word for the common pea (*Pisum sativum*), but that brought to Liverpool is different from the ordinary pea of this country, and resembles a lentil more than a pea. It is imported into Glasgow from India in large quantities, I believe, mostly as ballast, and has, I am told, not only been given to horses, but ground and mixed with various cakes for cattle, and with many fatal consequences.

However, in the outbreak at Liverpool, it appears that horses commenced to die very suddenly some time after the owners had commenced to use the mutters, and for several weeks after they had discontinued to use them they still died. The symptoms were roaring, hæmorrhage from the nose, great prostration, swelling of the throat, succeeded in many cases by sudden death. Other horses, however, lived a considerable period; but none recovered in which roaring had become pronounced.

I saw them in March, and found two dead on my arrival—one having only been dead a few hours; and from the blood of which I obtained the bacilli shown in the drawing.

On examining the food everything was found to be of the best and cleanest quality; but the Indian mutters were very dirty, dusty, and mixed with the excrement of rats; and from this dust bacilli identical with those found in the blood were cultivated; but in no instance did I succeed in obtaining bacilli from the interior of the grain. From this it may be inferred that the microbes were in the dirt surrounding the grain, but not in the mutters themselves, and that by proper washing and cleaning they might be a safe diet.

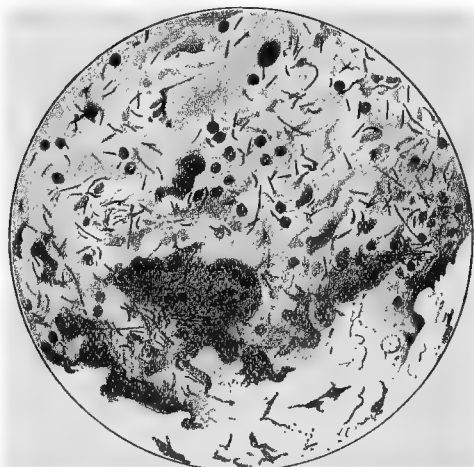


FIG. 28.

● Blood corpuscles.

Bacilli of various lengths, but about  $\frac{1}{1000}$  inches in diameter. These bacilli seem to differ from those of splenic fever, being rather smaller in diameter, and, so far as my observations go, multiply by fission only, not developing spores. This, however, requires confirmation.

This, however, requires further investigation, as animals have died on the Continent presenting similar symptoms to those at Liverpool after being fed on the legumen *Lathyrus sativus*, a bitter legumen; but whether from a vegetable poison contained in the legume or from bacillary growths has not yet been determined.<sup>1</sup>

In animals which have white skins, or where an eruption

<sup>1</sup> Further experiences of the effects of *lathyrus* poisoning have led me to conclude that the hæmorrhagic engorgement of the throat and the epistaxis were accidental complications due to the tainted state of the food in these particular cases seen at Liverpool. The effects of feeding with *lathyrus* are now well known, and will be referred to in the chapters dealing with Dietetic Diseases.

takes place in parts of the body void of hair or wool, red, brown, violet, or mulberry spots of bloody effusion are seen. These spots are independent of tumours and extravasations, and sometimes exist on the surface of the tumours. They are most commonly seen in the pig and sheep.

Ecchymoses are seen on the visible mucous membranes. When the fever progresses slowly these spots unite by confluence, and surround, notably in the pig and sheep, the whole body. Some of them become crepitous and emphysematous; others take the form of tumours, passing rapidly to the state of gangrene. In addition to these eruptions, there is often a soreness and swelling of the throat, infiltration of the upper end of the trachea, and a discharge of a lymph-like material from the nostrils, constituting what is termed *gloss-anthrax*, common in the pig which was fed upon anthrax flesh. It is also seen in the ox as well as the horse.

Charbonous tumours are generally of a black-brown colour. The phlyctenæ are filled with a brown liquid, which is very irritating; it sweats on the surface as drops of cold serosity. The tumours have little tendency to suppuration, and speedily become gangrenous. If these symptoms are added to those furnished by the mucous membranes, no difficulty need be felt in diagnosing charbonous tumours, phlegmonous and gangrenous.

*Terminations.*—When the tumours resist mortification, they terminate by delitescence, by suppuration, and by metastasis.

*1st. Delitescence.*—The morbid products are effused, and constitute tumours, which are reabsorbed and expelled by the excretory organs. Examples of this kind of termination have been observed by veterinarians in Africa.

*2d. Suppuration.*—When the tumours progress in a slow manner, indurating gradually, suppuration may be brought about by therapeutic and surgical means. Resolution is slowly induced, and it is not without pain that the necessary suppuration can be provoked. The animals remain poor and unthrifty, and often in the horse glanders and farcy conclude this morbid state.

*3d. Metastasis.*—The amelioration of the symptoms which succeed the development of tumours is not often lasting. The products are reabsorbed and carried anew into the circulation. This unhappy crisis is announced by the reappearance of all the symptoms proper to charbonous fever; their succession is so rapid that death may take place in from eight to ten hours.

## SYMPTOMS OF ANTHRAX FEVER IN HORNED CATTLE, APOPLECTIC ANTHRAX, SPLENIC APOPLEXY, SPLENIC FEVER.

The symptoms in the ox are very analogous to those in the horse. The ox suddenly goes off its feed ; rumination is suspended ; there are rigors and tremblings ; partial sweats bedew the body, which is alternately hot and cold ; the temperature rapidly rises, until in many cases it may become as high as 108° F. in the course of a few hours. After the onset of the disease this symptom should be looked upon as of much value in differentiating this disease from most other diseases. The dorso-lumbar region is excessively tender to pressure, and when it is the seat of the tumour, very acute pain is thus caused ; the gait becomes staggering, and the animal rapidly exhausted. A recumbent posture is almost constantly maintained ; the animal will now and then attempt to rise, but will rarely succeed in doing so. When standing, the back is arched, the legs stiff and rigid, but the standing posture is not long maintained. The animal looks towards its flank, falls into convulsions, and expels, without much effort, soft and bloody matter by the anus. The heart beats with violence against the thoracic walls ; the pulse is small, rapid, irregular intermittent, and sometimes double ; the conjunctivæ red, injected, and reflect a blackish-red tint ; the respiration is panting and plaintive ; there is tympanites of the abdomen ; the tongue is bluish-red, and the mouth filled with mucus ; blood escapes from the nose ; the eyes are sunk in their orbits, and tears flow over the cheeks. The areolar tissue of the back and sides becomes crepitous to the touch, and the animal dies during a convulsive exacerbation, or during the succeeding calmness. In some animals the excitement is so great that it is dangerous to go near them. The rapidity with which the symptoms succeed each other is variable, death taking place in the space of from a few minutes to even three days.

In cattle above two years old, particularly milch cows, the local lesions are often confined to great congestion of the spleen, and to a lesser extent of the liver and mucous membrane of the intestinal canal. In other, but rarer, instances, the engorgements may be in the lungs, and should the animal survive for some days, decomposition of the extravasated blood is estab-

lished, as expressed by fœtor of the breath, the decomposed extravasated blood being absorbed into the circulation, causing death by septicæmia. I have also seen this condition in a sucking calf.

In true splenic apoplexy the spleen is often much enlarged, broken down in structure, and its capsule distended with a mass of tar-like blood. If one end of the organ be elevated, it will be seen that the blood will gravitate into the most dependent part, showing that the splenic tissue is disintegrated, and that the blood is more or less fluid.

Death from splenic fever is very sudden ; in many instances an animal seen a few hours before apparently in good health is found dead, death having apparently occurred without a struggle. If, however, the disease is not so rapid in its course, it may be noticed that there are various alternations in the symptoms ; in some instances an animal will be unwell for several days, suffering from a remittent fever ; one day very ill, with rapid, feeble pulse, hurried and painful breathing, red and injected eyes, hot mouth, irregularity of the bowels, and redness of the urine. After continuing for some hours these symptoms may subside, and the animal commence to eat and ruminate. The febrile symptoms, however, often return, and in the end the sufferer too often succumbs.

In another form of anthrax without external tumours, the most prominent signs, in addition to the general disturbance, are severe colic and the passage from the bowels of quantities of dark coloured blood ; and the disease is then denominated *enteric* or *abdominal charbon*, the *post mortem* characteristics being congestion of the intestinal mucous membrane, more particularly of the small intestines, which are covered with petechial spots, with incipient ulceration in their centres, extravasation of dark coloured blood into the canal, and very often extravasations into the sub-lumbar areolar tissue ; the fatty mass surrounding the kidneys being loaded with extravasated blood, in a disintegrated, broken-down, tarry, semi-fluid condition, or covered with petechiæ.

#### SYMPTOMS IN SHEEP.

In this country anthrax in sheep assumes the enteric form, but on the Continent of Europe splenic apoplexy seems to be the form by which they are usually attacked.

Braxy in sheep—an anthracoid disease—is a form of septicæmia, simulating anthrax in its *post mortem* appearance. See Septic Diseases.

The other form of charbonous fever in the sheep, not commonly met with in this country, but which seems common on the Continent, is splenic apoplexy, the symptoms of which are similar to those witnessed in the ox. Indeed it may be mentioned that the malady in all its forms may be accompanied by splenic congestion.

ESSENTIAL CHARBON, OR THAT ARISING FROM INOCULATION—  
MALIGNANT PUSTULE (PUSTULA MALIGNA) IN MAN.

*Definition.*—Implanted on some uncovered part, the organism produces in the first instance a redness like the bite of a gnat, and afterwards a minute vesicle. A peculiar form of gangrenous inflammation is excited, which rapidly spreads from the point first affected to the neighbouring tissues. Hardening and blackening of the part is so extreme, and death of the tissue is so entire, that the part cracks when cut with a knife. No pain attends the incisions; crops of secondary vesicles form round an erysipelatous-like areola, chains of lymphatics become inflamed, the breath foetid, and death follows, amid all the indications of septic poisoning.—(Dr. WM. BUDD.)

Such is the disease in man, and its identity with charbon has been satisfactorily proved by the fact that, when contracted by man, it has been communicated by inoculation to the lower animals. Malignant pustule in man is concurrent with charbon in cattle, &c., and is a result of direct inoculation. Other cases occur in which the exact vehicle of the poison cannot be identified, but these cases have all this significant peculiarity, that the disease is always *seated on some part of the person which is habitually uncovered.*—(AITKEN.)

*Propagation.*—The disease may be communicated to man in the following ways:—By direct inoculation, as in the case of butchers and others employed to skin the carcasses of animals which have died of charbon, the poison finding access by means of the skin or hands or arms of the operators; by means of the skin or hair of animals dead of charbon; and there are many examples related by Dr. Budd which clearly prove that the virus, when once in a dried state, may retain its virulence for an inde-



finite period of time. Trousseau relates that in two factories for working up horse-hair imported from Buenos Ayres, and in which only six or eight hands were employed, twenty persons died in the course of ten years from malignant pustule. The disease may be communicated by eating the flesh of animals killed while affected with it, as also by using the milk and butter of affected cows.

#### ANTHRAX IN THE PIG.

The observations of Klein having proved that the very fatal disease amongst pigs known as anthracoid erysipelas, the blue sickness, pig typhoid, &c., differs in many particulars from anthrax; the varieties witnessed in the pig are reduced to anthrax fever, gloss-anthrax, and anthrax with tumour. Anthrax fever is as rapidly fatal in the pig as in other animals, destroying life in a very short time, with but slight manifestation of sickness, killing by shock. In milder cases, however, there is loss of appetite, sudden prostration, sullen appearance, hanging ears, painful and haggard expression, vomition of a coffee-coloured fluid, continual convulsions, paralysis of the extremities, rapid alternations in the heat of the body, highly injected mucous membranes, and generally terminating in death.

*Gloss-Anthrax—Malignant sore throat—Anthracoid angina—*This form of anthrax is most commonly seen in the pig when it has fed on the flesh of other animals which have died of the malady. It is rapidly fatal, the throat swelling enormously; the pharynx, larynx, tongue, &c. becoming enormously swollen and gangrenous; an exhaustive diarrhœa, with great tenesmus and discharge of blood, often appearing prior to death.

I have known of several instances in which pigs have died in great numbers after having eaten of the flesh and offal of cattle which had died of quarter-ill and splenic apoplexy; and Mr. Borthwick, V.S., Kirkliston, has told me an instance which occurred in 1872, in which twenty-five pigs died in two days after having eaten the flesh of a bullock which had died of splenic apoplexy.

#### ANTHRAX IN POULTRY.

Charbonous fever is announced in poultry by the following symptoms:—No appetite; feathers ruffled; walk difficult and staggering; foetid diarrhœa; great prostration; dragging of the

wings; turgescence or blackness of the conjunctivæ; excessive sensibility of the extremities. The fowls squat, and do not look for a perch; the bill and comb become black; tumours, or red spots, which soon become black and gangrenous, form on the palms of the digits, and the animal dies in convulsions.

#### PATHOLOGICAL ANATOMY OF ANTHRAX.

*Post mortem* examinations disclose very manifest lesions, which explain the rapidity with which putrefaction has taken place in the tissues.

(1.) *Exterior*.—A short time after death the whole body is tumefied and disfigured from excessive formation of gas in the areolar tissue. The abdomen is considerably swollen from gas in the gastro-intestinal organs, and blood-clots escape from the nose and mouth; the rectum is reversed, and looks like a black tumour, from the centre of which gas and tainted liquids escape.

On different parts of the body, notably on the skin deprived of hair, red or mulberry spots, sometimes interspersed with yellow ones, are found.

After being opened, the body emits a very foetid odour.

(2.) *Skin, cellular tissue*.—In incising the skin crepitation is heard, which results from the disengagement of gas accumulated in the subcutaneous areolæ; black and liquid blood escapes from the parts cut with the knife.

The skin on the tumours and engorgements is found semi-detached, and its internal surface presents spots of infiltration corresponding to those seen externally, and the tainted skins are at these spots without consistence, and depreciated in value.

The areolar tissue is the seat of blood and sero-albuminous infiltrations of yellow, red, or black colour, which extend into the interstices of muscles and to deep-seated organs.

(3.) *Muscular tissue*.—Yellow infiltrations on the surface, and to a less degree in the bed of the muscles, in the form of lines which put the fibrillæ in relief. The muscular system generally is impregnated with black blood, which communicates this colour to the whole frame.

The normal adhesions of the muscles to the bones, tendons, and aponeuroses are so relaxed that one may detach them without much effort.

In the thickness of the muscular substance black spots are found from the escapement of blood. These morbid alterations are most marked in tumours and charbonous engorgements, constituting masses of serosity of a citron colour, which raise the skin, infiltrate the subcutaneous areolar tissue, and penetrate deeply into the organs of the body. Infected gas is disengaged from the surface of the divided tissues; here and there portions of the tissues are decomposed.

It is a remarkable and important peculiarity that in these vast engorgements no trace of inflammation can be found, no plastic material, no injections, no vascular arborizations, which characterise the inflammatory process.

M. Delafond has recognised the following microscopic particulars:—

(1.) That where tumours are situated the tissue is penetrated by blood globules deprived in part of their colouring matter.

(2.) That the red colour of the tissue is due to the escape of the hæmatin from the red globules.

(3.) That the serosity of the infiltrations is fibrino-albuminous.

(4.) That this serosity is associated with a great number of very little globules, abnormally indented on their surfaces.

(5.) That the areolar plates, in which this serosity is deposited, are slightly opaque from coagulation of fibrin.

The morbid lesions of the solids are evidently a consequence of the primitive alteration of the blood.

The large vessels—the aortæ, venæ cava, vena porta, the cavities of the heart—are filled with fluid blood reflecting a strong dark violet tint, presenting no signs of coagulation. The walls of the vessels and of the heart possess a red colour, which resists washing, showing that the colouring matter has penetrated the tissues.

These alterations explain the formation of black spots, of effusions, of bloody extravasations, and of serous citron infiltrations on the surface and in the depths of the organs. At the same time the deficiency of fibrin renders the fluid incoagulable both during life and after death. The heart is flaccid, soft, and covered with bluish spots. The blood in both sides of the heart is black, liquid, and more abundant in the right than in the left side of that organ. When exposed to the atmosphere it retains its black colour, or is but very slightly reddened. Some deny that it changes colour at all.

The pericardium is covered with ecchymosed spots. The pericardial fluid reflects a red tinge, more or less marked, according to the time which has elapsed between death and the autopsy.

(4.) *Lymphatic system*.—The ganglia of the cervical region, of the thoracic cavity, of the dorso-lumbar and inguinal regions, are always diseased. They are much increased in volume, ecchymosed, yellow or red, soft, impregnated with a great quantity of serosity, and a citron-coloured infiltration, and are easily pulped by pressure between the fingers.

These characters are found in a degree more or less marked in the lymphatic ganglia of all the organs; on minute dissections being made, cords and little nodules are seen of a yellow colour, and of various forms and sizes, and the lymphatic vessels, especially those that have the tumours, are much distended.

(5.) *Digestive apparatus*.—The peritoneum, epiploon, and mesenteries are irregularly covered by ecchymosed spots. The peritoneal cavity contains serosity of a deep colour, mixed with blood-clots. The large veins which follow the convolutions of the intestines have a bluish aspect, and are distended with very black blood. In the mesentery, and above all in the sub-lumbar region, charbonous tumours of various dimensions are found. They are very common in horses which have died of charbonous disease without external eruption.

The tumours are formed by a mass of very black blood of a syrupy and gelatinous consistence, and by an infiltration of serosity of a yellow colour. They are situated in the fatty masses that surround the kidneys, the pancreas, the posterior vena cava, the sublumbar ganglia, and between the folds of the mesentery, immediately above the convolutions of the intestines. The intestines present exteriorly a red colour, more or less marked, following the extravasation and escape of blood. The lesions found in the interior of the canal vary from a simple red discoloration to a dark congested state of the mucous membrane. When washed, the villi are seen congested and augmented in volume. In other cases blood is extravasated and fills the intestinal conduit, mixing with the alimentary matters. It rapidly decomposes, and emits a bad smell. Under the mucous membrane a yellow infiltration is found, extending into the sub-peritoneal areolar tissue. Peyer's patches do not

present any characteristic alterations. In some cases the mucous membrane surrounding them has been seen to ulcerated; and in animals of the bovine species the glands Brunner have been seen to be augmented in volume.

(6.) *Spleen*.—This organ is the seat of very remarkable lesions and in virtue of the general constancy of their character, they may be considered as the most conclusive expression of the existence of charbon. Its volume is often double, triple, quadruple; it is larger, longer, and thicker, and its external surface has a livid blue or black colour. The enlargement is sometimes uniform, sometimes irregular, and is formed by a mass of blood distending the splenic capsule, which occasionally becomes ruptured, giving exit to thick, black blood.

When cut, black incoagulable blood escapes from the incision. On pressing and washing the tissues, the putrid matter is removed, and the fibres left are of a reddish-black colour.

(7.) *Liver*.—Augmented in volume, with the appearance having been boiled. Its tissue is friable, easily cut, and from the cut surfaces great quantities of black blood escape.

(8.) *Respiratory organs*.—Some amount of serosity in the chest, of a muddy and slightly red colour. The costal and pulmonary pleuræ are covered with black spots. In the subserous areolar tissue there is a yellow citron infiltration, which is continued into the interlobular areolar tissue. Gas is developed in the subcutaneous areolar tissue, and brown and black spots are seen throughout the lung tissue and on the respiratory mucous membrane.

(9.) *Nervous system*.—The meninges of the brain and spinal cord are covered with black spots. The venous sinuses are filled with a very liquid blood, and in places yellow infiltrations are observed. The cerebral substance is ecchymosed, and clots of blood are seen on its surface.

The ganglia of the great sympathetic nerve are large, soft, and infiltrated.

(10.) *Urinary apparatus*.—Like the liver, the kidneys are augmented in volume. They are of a brownish colour, easily broken down, and the blood with which they are filled escapes when they are squeezed.

A consideration of the lesions found *post mortem* leads us to one conclusion—namely, that the alterations seen in the solid

are due to a profound modification which has been established in the physical and chemical characters of the blood.

In the horse more particularly, the jelly-like yellow and sero-hæmorrhagic infiltrations are encountered nearly everywhere in the body where there is loose connective tissue, chiefly in the retro-pharyngeal and laryngeal tissues, along the course of the great blood-vessels of the neck, in the mediastinum, peritoneum, and about the kidneys. The corresponding lymphatic glands, especially the mesenteric, show sero-hæmorrhagic infiltrations, are considerably enlarged, and here and there in a state of incipient gangrene. The intestinal lesions in the horse are for the most part not so diffusely spread; but still upon the mucous membrane, which is in a catarrhal state, cedematous, and sprinkled with ecchymoses, there are found well pronounced carbuncles, which are the seat of more or less superficial sloughs. The intestinal contents are likewise often bloody and thinly fluid. The large glands, liver, and kidneys as a rule are swollen, the parenchyma cloudy, succulent, and full of blood. In the blood, besides the presence of bacteria, the white corpuscles are found to be in considerable number. The red corpuscles for the most part are of lessened consistence, and manifest a tendency to cohere in little heaps. On microscopic examination of the carbuncles in the intestines, as well as elsewhere, and of the jelly-like hæmorrhagic effusion into the connective tissue, there are found in the capillaries, which are considerably diluted, besides a massing of white blood corpuscles (cellular cedema), numerous bacteria, and a finely granular mass, consisting partly of metamorphosed blood detritus, and partly bacterial germs. The thickness of the blood is due to its being deprived of water by the dropsical, jelly-like, and cedematous effusions. The dark colour is caused by carbonic acid poisoning.—(BOLLINGER.)

#### TREATMENT.

When the symptoms of the disease, in any of its forms, are fully developed, but little good can be effected by medical or surgical interference. Some few cases may, however, be amenable to treatment.

The blood is fluid, dark, imperfectly coagulated, containing the constituents which unite to form fibrin in a diminished quantity,

or in an altered or modified condition. How, then, is the viscosity of the blood to be restored? Direct experiments upon the blood removed from the body, and the treatment of hæmorrhagic diseases, have very satisfactorily shown that the chlorate of potash has a peculiar effect in this direction, and it is upon this substance that dependence must be placed. For young stock the following draught is to be administered:—

R. Pot. chlor. ℥iii.

Aquæ,      Oi.

M. and give three times a day.

For a full-grown animal an additional drachm may be given with safety; but if given in large doses intestinal irritation is apt to be induced.

On account of its well-known antiseptic properties, carbolic may prove very useful in the treatment of anthrax; indeed, I understand that Principal Veterinary Surgeon Collins has prescribed its internal administration with marked effect in India. It may be given largely diluted, two or three times a day, in doses proportionate to the species and size of the animal under treatment. M. Decroix, P.V.S., French Army, recommends the borate of soda, 100 grammes daily, dissolved in water, and given as a draught to the horse.

Purgatives generally do harm, and for the reason that there is some congestion of the intestinal mucous membrane.

During the course of treatment of charbon it is important that special regimen be used. The animal should be well nourished. Food easily digested and of good quality should be given. Tonic medicines, as preparations of iron, gentian, and decoctions of aromatic plants, may be used at the time of convalescence.

The prevention of charbonous fever is a matter of great importance. Some writers affirm that it is more commonly met with since the introduction of artificial manures, and that it is due to the presence of the nitrites conveyed from the soil to the blood in the water, herbage, turnips, &c. I think that this view is not established, as the malady is met with where artificial manures are not at all used. If it were solely due to the nitrites or to any of the salts contained in artificial manures, its existence would have been unknown prior to the introduction of such manures. It is a fact, however, that when animals are poisoned with the nitrites, such as the nitrite of amyl, that the blood

assumes a chocolate colour (Dr. A. Gamgee) ; and it is also a fact that certain diseases are more prevalent on rich than on poor soils.

*Preventive Treatment.*—It is supposed by many veterinarians of experience that the introduction of setons is a very effectual preventive of charbon, and their operation is explained by the fact that in inflammations, artificially produced or otherwise, the coagulating properties of the blood are increased, as well as the power of phagocytosis. The quantity of fibrin discoverable after the blood is removed from the body is appreciably increased. The safest and quickest method of creating an artificial inflammation is by inserting a seton in the breast—the seton to be deeply inserted, and in order to increase its irritating properties it should be dressed with blistering ointment. If the disease has appeared amongst a herd before the veterinarian's attention is directed to any method of prevention, in addition to setons, the chlorate of potash is to be given, mixed with the animal's food or water for several days, the diet in the meantime being restricted, and all circumstances likely to promote the malady fully inquired into and removed.

It is also essential, not only as a means of prevention, but after the actual occurrence of the malady, that the excretory organs be kept performing their proper functions, and for this end gentle aperients and diuretics should be employed. I have already pointed out the danger of administering drastic cathartics, but there is no reason why the bowels should not be gently opened by laxatives, such as linseed or castor oil. If during the course of the malady it is thought necessary to employ stimulants to rouse the flagging animal energies, the preparations of ammonia are to be avoided, as they cause the blood to lose its coagulating properties, both within and without the body ; and, as already stated, an excess of ammonia already existing in the blood, alcoholic stimulants are to be given in preference. Acting upon this conclusion, I at one time administered acid preparations, and with some success, but a larger experience has taught me that the chlorate of potash is superior to all other medicines.

The flesh of animals which have died or have been killed whilst suffering from the disease should not be used as food,



as it is apt to cause death, and the milk of affected cows has proved fatal to man.

Seeing that the curative treatment of anthrax in the lower animals is futile, and in fact attended with danger not only to the attendants, but also in facilitating the spread of the disease, it is recommended that affected animals should be immediately destroyed and extreme measures taken in the matter of prevention by adopting segregation and disinfection. The preventive treatment is now supplemented by the administration of both sera and vaccines. The advantage of the former is that their effects are demonstrable immediately, but are not lasting—*i.e.*, they do not endure for more than about ten days, whereas by vaccination, although attended by a certain amount of risk, the immunity conferred is for a period of ten to fourteen months.

Preventive serum is obtained by immunising animals and increasing their immunising power by the repeated subcutaneous and ever-increasing doses of virulent culture. Blood from such animals is withdrawn, serum obtained by defibrination and centrifugalisation or by sedimentation, under strictly aseptic precautions, and then mixed with 25 per cent. lysol. This serum in proportionate doses may be used either as a preventive or a curative.

Vaccines are obtained from the pure cultures of the organism. Pasteur found that the inoculation of bacilli whose capabilities of sporulation had been destroyed did not cause death, but gave an active immunity which was more or less permanent. In order to obtain a culture of organisms incapable of sporing, the organisms must be grown at a high temperature; and to render the vaccination more complete two inoculations are made, the one with strong, and the other with weak, vaccine. The weak vaccine is made by growing an anthrax culture at  $42.5^{\circ}$  C. for twenty-four days; a fixed quantity of this will kill mice, but not rabbits or guinea-pigs. Ten days after the administration of this weak vaccine a dose of the strong is given. This is obtained by growing cultures of anthrax at  $42.5^{\circ}$  C. for twelve to fourteen days. A fixed quantity of this will kill mice and guinea-pigs, but not rabbits, and is used to strengthen the cattle's immunity and render that protection more complete and lasting. The objection to

this method is, so it is said, that one is spreading the disease or is liable to raise infection by its use, as one never can tell if a culture, so prevented from sporulating, may not recover that power. There is also in the course of its administration a certain percentage of deaths, although small. In the use of vaccines it is always advisable to use *killed* cultures, and thus to obviate such dangers, but it has been found that a killed culture has not such advantageous powers as a weakened one. A combination of serum and vaccine has been advocated, as the serum gives immediate immunity and the vaccine the subsequent permanent protection; moreover, the one operation is sufficient, as the serum will so give the temporary protection that it is possible to administer the stronger vaccine at once. The two are inoculated separately and at different sides of the body, as in rinderpest. Here, again, the danger of infecting clean areas is evident, although it may be said that when such operations are necessary the disease already exists, and that the remote chance of spreading a fresh infection is not worth consideration, and would not matter anyhow.

In the Argentine and other places benefit is said to be obtained by the use of the two vaccines, but for the reasons given the method has not found favour in England.

#### QUARTER-ILL, BLACK-LEG, OR CHABERT'S DISEASE.

Quarter-ill, black-leg, strike, symptomatic anthrax, quarter-evil, sarcophysema hæmostaticum bovis, infectious emphysema, are a few of the numerous names applied to a specific disease affecting cattle and sheep, and occasionally horses and pigs, and due to the presence of an organism, the *Bacillus Chauvæi*, also called the *Bacillus sarco-physematos bovis*.

Quarter-ill was, until recently, supposed to be an external manifestation of splenic fever (anthrax), that in young cattle anthrax was always thus developed, whereas in older ones the disease was located in the spleen.

This disease is fortunately not nearly so common in Scotland as it is in England, France, and other countries on the Continent; but owing to better drainage of the land it is less common than formerly, even in England.

It occurs much more frequently in young than in old animals,

as is well illustrated by the following record by M. Hess of 989 cases :—

374 cases in animals aged between 6 and 12 months.

439	„	„	1 and 2 years.
83	„	„	2 and 3 „
65	„	„	3 and 4 „
10	„	„	4 and 5 „
18	„	„	5 and 6 „

Not only is it less frequent in old animals, but it is also less fatal to them. Animals affected under two years old almost invariably die, but animals over that age frequently recover: so much is this so, that many affirm all animals over three years of age to be free from danger of contracting this disease.

But this conclusion, however, must not be implicitly believed in, as the following statements by Hess show there is even danger though the animals are beyond the stated age.

Out of 36,000 animals, from six to twelve months old, 350 died of the disease; of 13,000 animals, from one to three years old, 500 died; and of 135,000, from three to six years of age, 120 died; giving us the following percentages of total mortality—

Between 6 and 12 months, .972 per cent. died.

„	1 and 3 years,	3·845	„	„
„	3 and 6	„	·08	„

The disease is never, or hardly ever, seen in calves under six months old, unless they are fed on a diet which is not an exclusively milk one.

Quarter-ill, like many other diseases, has periods at which it is rife in the country, and other periods when it seems to be dormant.

It is most frequently seen when animals are changed from one pasturage to another, or from one condition of living to another, more particularly when the change is from poor feeding to rich pasturage. The disease is also most prevalent in low-lying pastures, and when there is rain and humid heat, though it may occur on any lands and in all kinds of weather.

*Symptoms.*—The symptoms of the disease are usually mostly as follows, but occasionally the initiatory ones do not indicate it very clearly, and may readily be taken for those of some other affection, and perhaps one which may be cured by treatment, and so consequently is apt sometimes to mislead even

the expert:—Loss of appetite, dulness, listlessness, cessation of rumination, harshness and staring of the coat, elevation of temperature, rigors and local tremblings, coldness of the extremities, then lameness or stiffness when moved, arching of the back, and on examination of the skin a tumour is found forming under it in some part of the body. It may be on the head, neck, shoulders, dewlap, loins, genital organs, or mammary gland, but by far most frequently in either the shoulder or the loins.

M. Hess has observed that the tumour forms more often on the right side of the animal than on the left, but he can give no reasons why this should be so.

The tumour is found in regions which abound in muscular tissue, and where the connective tissue is loose, and seldom in the region of joints and tendons, and where the tissues are firm.

These tumours are ill-defined, and have no limiting membrane. Externally they may not appear of great size, but when carefully examined they are found to extend deeply into the sub-jacent tissues.

At first hot and painful to the touch, they rapidly become cold, insensitive, and dead in their centres, and when then handled are found to crepitate or crackle, due to the presence of evolved gases under the skin; their peripheries extend and penetrate into the surrounding parts until they attain enormous dimensions.

If incised, they discharge a dark-coloured and foetid, acid fluid, succeeded by a flow of frothy, citron-coloured serosity.

As the disease progresses, the tumour or tumours enlarge, gases are evolved beneath the tissues, the animal evinces great distress, the breathing becomes greatly hurried, the temperature rises to a great height, the pulse beats at 120 to 130 per minute, feebly and intermittently; the expression of the face becomes haggard, tympanites (hoven) ensues, fæces are passed involuntarily, the animal falls, becomes unable to rise, is attacked by fits, and either dies during one of these, or immediately after its cessation.

In the last stages the temperature falls below the normal, and decomposition sets in even before death.

There are, however, cases which do not exhibit the ordinary symptoms to commence with. In some the disease begins as colic, or some digestive derangement; and in others there may be lameness and stiffness, but no appearance of a tumour

anywhere. On *post mortem* examination, the animal is found to be enormously swollen, due to the evolution of gases into the stomachs, intestines, and in the tissues under the skin. Bloody froth is seen issuing from the mouth, nostrils and anus. On cutting into the carcase, gases of a sour odour escape, and are often accompanied by spurts of dark blood or yellow serosity.

The tumours are found to be black in their centres, and the muscles in their immediate neighbourhood to have the same colour. The farther we proceed from the centre of the tumour the lighter the colour becomes, till, instead of being black, it is black and yellow streaked, then pinkish, and then surrounded by a citron-coloured portion.

In some cases the tumour is found in the muscles under the shoulder—in others in the diaphragm and in many varied situations, but these are rare compared to those found in the loins and withers.

*Disposal of carcase.*—It is the practice in some parts of England to slaughter cattle affected with the disease, to prepare them for human food by carefully removing the discoloured portions, and sending the rest of the carcase to market. We are opposed to this, and consider that this disease should be dealt with by the authorities, under the “Anthrax Order of the Contagious Diseases (Animals) Act.”

Quarter-ill is not capable of infecting man, and if the diseased parts be removed there would, in all probability, be no danger in consuming the flesh; nevertheless, the rapidity with which putrefaction sets in in such cases renders it extremely undesirable that such meat should be exposed in the public markets, and it is recommended that animals which have died from the disease should be destroyed.

The blood of an animal affected coagulates readily, and hence is quite different from that of anthrax (splenic fever).

*Cause of the Disease.*—The cause of this disease is an anaërobic germ, which when in connective tissue and outside blood-vessels propagates itself with wonderful activity, and in so doing causes destruction of the tissues in its neighbourhood, the formation of the tumours, and an excessive evolution of gases of a very foetid odour, and ultimately death.

This germ is smaller than the true *Anthrax bacillus*, measures from  $\frac{1}{5000}$  to  $\frac{1}{4250}$  inch in length, and from  $\frac{1}{5000}$  to  $\frac{1}{2500}$  inch in breadth. It is found in the tumour, and the exuded fluids contained in and about it, in large numbers, is scarce in the blood itself; it varies in shape,—cylindrical when the contents are homogeneous, club-shaped when sporulated, but when the spore is central the bacillus becomes spindle-shaped, and often contains a clear spot or spore at one end (known as racquet form), sometimes one at each end (known as spectacle form). It is motile, its movement being from side to side. Many of the rods contain spores, and have an irregular swollen appearance. If iodine solution be added, the germs stain a violet colour. They also stain with fuchsin, more particularly the spores contained in them, with careful technique—*vide* bacteriology works. They grow rapidly on glycerine agar and other cultivation fluids, liquefying gelatine, and evolving much gas, having an unpleasant sour odour.

The virulence in culture is easily lost and difficult to regain. It is, in fact, difficult to cultivate under the strictest anaërobic conditions, in a pure condition. Virulence is regained by inoculating guinea-pigs, and taking some of the resulting exudate and inoculating others, and so on, until it is lethal. As the disease affects muscular tissue, it is best obtained by inoculating directly into muscle, and by making a *post mortem* immediately after death; then the muscle is carefully excised and dried. The virulence is by this means unimpaired; and if the procedure be carefully carried out, a piece of such muscle may be kept for years and retain its virulence.

*Protective Inoculation.*—It has recently been demonstrated that when the germs are introduced directly into the blood-stream, and not into the connective tissues, immunity to the disease is caused.

To obtain the germs for protective inoculation the following is the method recommended by Messrs. Arloing, Cornevin and Thomas, and others, and found to be extremely successful:

From an animal just dead of the disease, take the blackest portion of the tumour, cut up into small pieces, mix with distilled water, then triturate in a mortar, squeeze through cloth, and filter through several folds of muslin which has

been previously wetted with water, and of this fluid inject from five to ten drops into the jugular vein by means of a hypodermic syringe, taking the very greatest care that none of it escapes into the wound. If the operation be carefully performed, the animal will have immunity conferred upon it.

In many, for two or three days, there is dulness and disinclination to feed, but this soon passes off, leaving no ill-effects.

But if the virus escapes into the wound, or if the animal has any bruises upon it, there we may expect to see the tumour of quarter-ill appearing, and death resulting.

Others, again, instead of using the virus direct, inoculate a guinea-pig subcutaneously, and cause the formation of a tumour containing germs, which are thus modified in their power, and after mixing with water, triturating, and passing through muslin, use it. Others, again, have used the desiccated virus prepared by drying in air portions of the diseased tissues. Cornevin, Arloing and Thomas's method of using the virulent tissue mixed with water and filtered as above stated, is the best, if it were not attended with such danger which renders it too impracticable.

The method now in use is by two vaccinations, which are prepared as follows: Strips of muscle characteristically affected, and uncontaminated, are dried in an oven at a temperature of from  $32^{\circ}$  to  $37^{\circ}$  C. When quite dried, it is powdered and mixed into a paste with distilled water. This is smeared on plates, and these placed in a thermostat for a first vaccine. These should remain in for six or seven hours, at a temperature of  $103^{\circ}$  C. For a second vaccine other plates are placed in a thermostat, and kept there for six or seven hours, at  $90^{\circ}$  to  $95^{\circ}$  C. The dried material must be carefully removed from the plates and measured. The dose of each vaccine is 1 centigramme, and the second vaccine given seven days after the first one. The vaccines may be mixed with water and given hypodermically, and near the extremity of the tail. Immunity is obtained in about a week after the second vaccine, and lasts a year.

Many other methods of preventing this disease have been in use from ancient times, and seemingly with success; setoning,

for instance, when performed on young animals not affected with the disease, but having been in contact with affected animals, almost invariably is said to protect the animal, for the time being at least. We think that the setoning itself does not protect, but that the dietetic and hygienic alterations which accompany it materially tend to stop its progress, and the credit is thus given to the wrong agent.





PLATE XII.



Geo. W. H. H. & Sons, Lith. Edin.

*From U.S.A. Bureau of Agriculture Annual Report.*

SWINE FEVER.—ULCERATED CAECUM.

*To face page 327.*

## CHAPTER XXVII.

### CONTAGIOUS DISEASES—*continued*.

#### HOG CHOLERA—SWINE FEVER—PNEUMO-ENTERITIS.

THE term swine fever is applied in this country to a contagious and infectious disease of a very fatal nature. The pathology of infectious swine diseases has been profoundly studied on the Continent of Europe and in America, and it has been found that the pneumo-enteritis of Klein, typhoid fever of Budd, hog cholera, blue sickness, measles, erysipelas, and intestinal fever are terms incorrectly applied to what was supposed to be one disease, but which has now been discovered to include three separate, although similar, pathological conditions; and the researches of Schutz, Löffler, Eggeling, Salmon, and others have brought to light that under these terms there are in reality three diseases, namely—*A.* Swine plague, also called *rouget de porc*, angina, petechial fever, &c., characterised by septic gastro-enteritis, hæmorrhagic nephritis, with congestion of the spleen, inflammation of the heart, liver, and voluntary muscles, induced by a very small immobile bacillus, stainable by Gram and Weigert's preparations. *B.* Contagious or infectious pneumonia, pleuropneumonia, having a tendency to pulmonary gangrene and caseous products, and arising from an ovoid bacterium of the bipolar staining, Gram negative, Pasteurella type. *C.* Hog cholera, diphtheria, pneumo-enteritis, swine fever, &c., characterised by an inflammation and ulceration of the glands, large intestines, enlargement of the mesenteric glands. The ulcerations of the glands of the large intestines are situated in the cæcum, immediately posterior to the ileo-cæcal valve, distinguished from swine plague by its slower development, its tendency to involve the pulmonary organs, and by differences in the specific microbes, which are motile, ovoid bacteria, aërobic and facultatively anaërobic, unstainable by Gram's

method, and but slightly coloured by Weigert's process. This is the disease usually found in Britain. Recent observations have brought to light the fact that infective material, when filtered through a porcelain filter, is still infective, though no organisms can be discerned microscopically. If this be so, it will place swine fever amongst the already long list of diseases due to an ultra-microscopic organism, and show that the organism at present considered the cause is only an accidental contamination. Further investigation is necessary.

*Definition.*—Swine fever may be defined to be a highly contagious and infectious disease, having a period of incubation, after inoculation, of about five days, at the end of which period there is elevation of temperature to  $104^{\circ}$  to  $106^{\circ}$ , succeeded by signs of general ill-health, and generally a rash on the skin.

When propagated by cohabitation, it appears, according to experiments performed by Dr. Lutton in America, that the disease took about thirteen days to manifest itself in healthy pigs amongst which diseased ones had been introduced.

The disease is very common in Great Britain, Ireland, America, and various parts of the world.

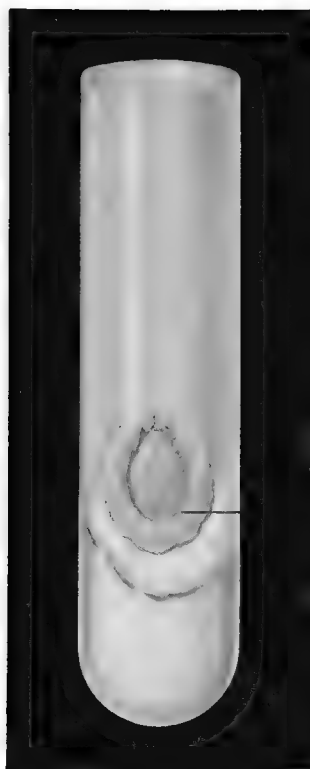
It prevails as an epizootic, and is the most fatal malady to which swine are liable; but pigs differ in their susceptibility to it.

*Causes.*—Like cattle plague, pleuro-pneumonia, &c., swine fever appears to arise from contagion and infection only; no amount of bad management, filth, want of drainage, nor decomposing food being sufficient of themselves to induce it. The microbe is a small ovoid motile bacterium, both aërobic and anaërobic, measuring  $\frac{1}{25000}$  to  $\frac{1}{12500}$  in length, and  $\frac{1}{75000}$  of an inch in breadth. Klein, however, states that it is due to a small bacillus which in cultures assumes a long, leptothrix-like filament, which develops spores which become free after the disintegration of the filamentous matrix; Detmer discovered a bacillus which he called *Bacillus suis*, and said it was the specific agent. The disease is transmissible by inoculation to the mouse and pigeon, and, according to Law (Cornell University), to the sheep and rat.

#### PATHOLOGY AND SYMPTOMS.

The symptoms are loss of appetite, general prostration, small and frequent pulse, hanging ears, sullen appearance, painful and haggard expression, watery eyes, conjunctivæ red and spotted,

PLATE XIII.



Ulcer-like Growth in  
Nutrient Jelly.

BACILLUS LUTEUS (SUIS).



dirty secretion about the eyelids, generally preceded by a red blush and red spots on the ears, the abdomen, and internal aspects of the extremities.

The reddened spots are at first hot and painful to the touch, but afterwards become cold, humid, and insensible even to the pricking of a pin. As the disease advances, tremblings and convulsions are manifested, the animal grinds its teeth, the flexor muscles of the limbs contract, and the animal stands upon its toes. These symptoms are succeeded by paralysis of the posterior extremities, or of the whole body, involuntary defæcation and passage of high-coloured and even bloody urine. The bowels are generally torpid at first; but the fæcal matters may be soft, and mixed with very black, fœtid blood, and thick, tenacious mucus. Diarrhœa, however, often sets in; the defæcations are then profuse and exhaustive; the breathing becomes catchy and convulsive, a painful cough is present; the convulsions become more aggravated, and may continue to the end, or the animal becomes comatose, till death closes the scene.

In some cases the first observable symptoms remain stationary for a period varying from twenty-four to forty-eight hours; then the surface of the body becomes burning hot, and very sensitive to the touch, notably at the sides and abdominal walls. If touched, the animal cries with pain, and to these signs are added tremblings, convulsions, grinding of the teeth, and tetanic contraction of the muscles; succeeded by rapid diminution of temperature. The conjunctivæ become brown; the eyes bleared; the tongue dirty, thick, and bluish; and the animal, extended on its litter, incapable of any regulated movements, succumbs in from twenty-four to forty-eight hours.

These symptoms are liable to various modifications, depending upon the intensity of the fever and the various localizations of the poison. In some cases, the virus seems to expend itself upon the serous membranes, inducing peritonitis or pleurisy; sometimes upon the mucous membrane, as expressed by bronchitis or broncho-pneumonic congestion and hæmorrhage, enteric congestion and ulceration, sometimes even to perforation or rupture of the bowel.

In many cases the animal is amaurotic, wanders to and fro, falls down, rolls and kicks, and seems to be in pain. Now and then it will rise from its bed and give a piercing cry, the whole body being involuntarily convulsed.

The condition of the blood differs in a marked degree from that of anthrax, being fibrinous, red in colour, and as a rule contains neither bacilli nor the contagium of the disease.

The experiments of Klein point to the conclusion that—" 1st. The fresh blood of diseased animals does not, as a rule, contain the virus, as it fails to produce the disease when introduced into a healthy animal.

" Four animals were inoculated (at different times) with the fresh blood of diseased animals. They remained healthy. When subsequently inoculated with virus-containing matter, they became smitten with the disease.

" In a fifth instance, however, fresh blood did produce infection. [And this same blood proved active after having been kept sealed up in a capillary tube for several weeks.] This blood was obtained from a very severe case with copious peritoneal exudation; in which were found peculiar, abnormally large, coarsely granular cells; the same cells were also present in the blood; so that it appears probable that the blood became charged, by absorption during life, with matter from the peritoneal exudation. This latter always contains the virus in an active state.

" 2. Experiments showing that fluid as well as solid lymph of the diseased peritoneum contains the virus in a very active state.

" Six successful inoculations with fluid peritoneal exudation.

" There is no difference of activity to be noticed between fresh exudation and one that had been kept sealed up in a capillary tube for several weeks.

" Solid lymph obtained from the peritoneal cavity of diseased animals, having been dried at a temperature of about 38° C., proves very active.

" 3. Experiments showing that parts of the diseased lung, ulcerated intestine, and also diseased spleen, contains the virus in an active state. Diseased parts of lung or intestine that were dried at a temperature of about 38° C., retain their virulence unaltered.

" In all cases of pneumo-enteritis, the trachea as well as the bronchi have frothy blood-containing mucous matter, possessed of infectious properties. It must be therefore supposed that the breath of a diseased animal is charged with the poison. On



account of the diseased state of the intestine, also the dung is to be regarded as infectious.

" 4. Experiments showing that infection is produced by co-habitation with a diseased animal, or by keeping healthy animals in a place whence a diseased animal had been removed.

" 5. Several experiments were made to see whether feeding healthy animals on matter obtained from the diseased organs (intestinal ulcers especially) produces the disease. The experiment was always attended with success, if a lesion-abrasion existed in the mucous membrane of the mouth or pharynx; this was usually the case when the matter had to be introduced into the mouth while the animal was being held by assistants.

" There were, however, two cases which appear to prove that the disease cannot be produced by simple feeding.

" This was unfortunately at a time when I was not as yet acquainted with the fact that in many animals the disease is of so mild a form that it can hardly be recognised in the living animal. I have not made any *post mortem* examination of those two animals.

" But since then I have made two other experiments, in which the virus was brought directly into the stomach, by means of an india-rubber tube introduced per fauces and œsophagus. In both these instances the animals became diseased, and their intestines were most conspicuously affected.

" From the last three series of experiments, we may conclude that the principal way in which contagion of pneumo-enteritis is carried out, is through the instrumentality of the air and the food.

" 6. This series comprises experiments to prove that the virus can be cultivated artificially—*i.e.*, outside the body of an animal; in the case of splenic fever it has been successfully done by Dr. Koch.

" The experiments are seven in number:—(a.) Two refer to cultivations commenced with fluid peritoneal exudation; (b.) In the five others the virus had been obtained by cultivation of dried lymph from the peritoneum of an animal suffering from the disease.

" (a.) The cultivation of the virus for the first two cases was carried out thus:—

" Fluid peritoneal exudation of a diseased animal had been

collected and sealed up on November 6th, in a capillary glass tube. On the following day there was present a small clot due to coagulation. A minute speck of this clot was removed with the point of a clean needle, and with it was inoculated a drop of fresh aqueous humour of a healthy rabbit. This drop had been placed on a thin covering-glass, which, after the inoculation, was inverted over a small "cell," made by fixing a glass ring on an ordinary glass slide. The covering-glass was fastened on the glass ring by means of a thin layer of pure olive oil. The preparation was then kept in the incubator for twenty-four hours at a temperature of 32° to 33° C. After this time it was used to inoculate a new drop of aqueous humour in a similar manner to the one just described. We will call this the second generation.

"This new specimen was placed in the incubator, and kept there at a temperature of 32° to 33° C. for another twenty-four hours. In the same manner a third generation was started, by inoculating a fresh drop of aqueous humour. After having been kept in the incubator for several days, it was used to inoculate two animals at different times. Both animals became infected with the disease.

"(b.) The other five experiments were carried out with virus cultivated from solid lymph of the peritoneum of a diseased animal. The lymph had been dried at 38° C. (see series 2). A small particle of dried lymph is crushed into fine powder. With a granule of this, a drop of fresh aqueous humour is inoculated in the same manner as above described,—first generation.

"After having been kept in the incubator for two or three days at a temperature of 32° to 33° C., it is used to inoculate a second generation, care being taken to use a trace only of the fluid part, and not to come in direct contact with the original granule, which may be still discerned in the preparation.

"The specimen representing the second generation is kept in the incubator for a day or two. It is then used to inoculate a fresh preparation,—third generation. And, finally, this is used for establishing a fourth generation. After having been kept in the incubator, a part of it is used for inoculating *two* animals, the inoculation being carried out at different times.

"Both these animals became affected with the disease. Another portion of this fourth generation was used to start a

fifth generation, then a sixth, a seventh, and an eighth generation. With this three animals were inoculated at different times, All three animals became diseased in due time.

“ In order to correctly interpret the results of this last (sixth) series of experiments, it is important to mention that inoculation with dried lymph, diluted far less than would correspond to the third generation in the last-named experiments, is followed by a negative result.”

#### MORBID ANATOMY.

In the majority of animals, the skin about the perineum, groin, belly, and neck is swollen, and of a diffuse red or bluish-red colour, and the ear lobes and the skin of the nose also red and swollen, whilst in some instances there are gangrenous patches of the superficial cutaneous structures. In many instances, however, this redness of the skin may be entirely absent, even after death ; but the longer the animal lives, the superficial structures become, as a rule, the more swollen, and the dependent parts of the ears become deep red, puffy masses, from the surface of which the epidermis peels off.

The blood-vessels of the skin are more or less filled with blood or plugged with fibrin, and around the vessels lymphoid cells are discoverable ; the sudoriparous glands are greatly distended, and often filled with blood charged with large coarsely granular cells containing large clear vesicular nuclei. The connective tissue of the corium contains fibrinous exudations and a yellow serosity.

The small intestines almost invariably, and the stomach more rarely, are congested and covered with spots of ecchymosis, both upon their mucous and peritoneal surfaces. The large intestines have always the most characteristic appearances. There are isolated or confluent, generally roundish, ulcerations at and around the ileo-cæcal valve, the rest of the mucous membrane being congested and studded with spots of ecchymosis. Klein says the whole large intestine down to the rectum contains ulcers ; in the cæcum they are confluent, and measure several inches, extending transversely as well as longitudinally ; while the whole remaining mucous membrane of the large intestine is much thickened, and in some parts the mucous tissue contains large accumulations of blood. The ulcers are of various

aspects. The following forms may be seen—very minute, well defined, prominent yellowish-whitish specks of the size of a millet or hemp seed; then somewhat larger, more flattened, prominent, circular or oval, yellowish patches (with which in one case the whole mucous membrane of the cæcum seemed quite covered) of the size of a hemp seed, up to about one-eighth of an inch in diameter; next, flat, circular, or slightly oblong patches, situated on the crust of a fold of the mucous membrane, in size from one-eighth to one inch in diameter, generally black or grey (from bile pigment), except a very conspicuous, and, I may almost say, characteristic prominent rim, which is yellow. The ulcer generally shows a pale central or eccentric disc, around which the rest of the ulcer is arranged as concentric rings. Between these flat ulcers with concentric layers, and those uniform, yellowish-white, prominent patches

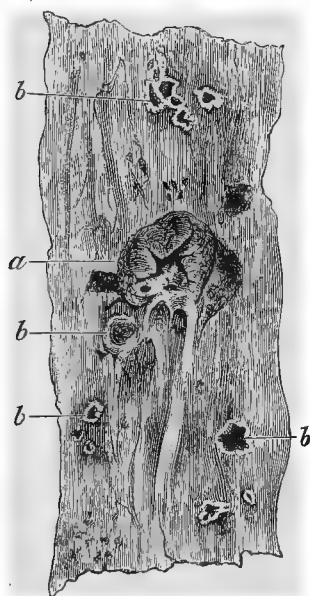


FIG. 29.—Portion of cæcum of American pig slaughtered at Liverpool, May 1879.

*a.* Ileo-cæcal opening.

*b, b, b.* Ulcers in various stages.

and nodules, there are all intermediary forms. This is easily understood, if it is borne in mind that as the latter increase in size, the central part is transformed into that black or greyish mass. Besides these, there are very often formed on the surface of the mucosa minute whitish specks, just perceptible by the unaided eye. In one single instance have I seen ulcerations of the lower part of ileum. This case had exceedingly numerous ulcerations of the large intestines.

The two ulcers of the ileum were quite different from the ulcers generally found in the large intestine, for they were oblong, deep pits, surrounded by a thick, prominent wall of swollen mucous membrane, very much the same as ulcers of human typhoid. But under any circumstances the microscope reveals a marvellous difference between the two diseases; for in the pig these patches, whether nodules or ulcers, have absolutely nothing whatever to do with lymphatic

follicles, whereas in human typhoid there is found in the first stage round, or generally oblong, prominent papulæ or patches of swollen lymph follicles, surrounded by swollen mucous membrane, after which stage the greater part of the swollen lymphatic patches dies, and is finally discharged as a slough leaving behind a pit-like excavation bordered by a well-defined fringe of mucous membrane, which is neither thickened nor indurated.

Next in importance to the intestinal lesions are those discovered in the lymphatic glands, which are congested, generally swollen, of a dark red colour, or infiltrated with blood, black, soft, and pulpy; those in connection with the intestinal canal are not only swollen, but infiltrated with a greyish-white opaque-looking matter.

The condition of the glands is sometimes very slight to the naked eye, there being merely swelling, and an apparent congestion; but even here the microscope reveals bursting of blood-vessels in the cortical tissue, and hæmorrhage into the lymphatic follicles and sinuses of that part, amounting in severe cases to total destruction of the adenoid tissue by blood. In severe cases the medullary sinuses, and partly also the medullary lymphatic cylinders, become filled with extravasated blood. In so far as this condition is similar to what is found in anthrax. "This state of the lymphatic system is very characteristic, and, combined with the disease of the intestine, is of paramount importance to the diagnosis."—(KLEIN.)

*Respiratory organs.*—In a large number of cases, the tongue, mouth, fauces, and pharynx are ulcerated, and sometimes gangrenous; the lymphatic vessels filled with micrococci; the lungs are generally congested in patches, with distinct mapping out of the lobes and lobules by œdema of the interlobular tissue, succeeded by hepatisation, at first red, subsequently opaque, or white specks or patches appear in the red substance, which, as they increase in size, become confluent. This is due to the fact that the bronchial tubes become gradually filled with a white, brittle, cheesy mass, progressing gradually from the finest ramifications on to the larger branches of the lobules and lobes. Finally the whole lobule is transformed into a discoloured, dry, hard, friable mass. The pleura of the corresponding parts is of course inflamed, being in some cases exceedingly thick, and

covered with false membranes. In severer cases, the greater part of one lung and portions of the other may be thus changed, and on the external surface there may exist smaller or greater ulcerations. Except in very slight cases, there is generally a certain amount of pleural exudation; and in severer cases the pleura contains a considerable quantity of a thick, offensive, yellowish or discoloured exudation. In some severer cases the pericardium is also inflamed, containing a large quantity of exudation, and its walls being much thickened by false membranes. The same is also the case with the peritoneum, this being in some cases hyperæmic in parts, or even covered with solid lymph and pus.

*The spleen and liver* are, in severe cases, dark in colour, enlarged, and the kidneys are sometimes changed, being hyperæmic and covered with petechiæ, particularly in the pyramids and underneath the capsule, which is easily stripped off.—(KLEIN.)

In the Privy Council Order dated 17th December 1878, which is termed also the "Typhoid Fever of Swine Order, 1878," it is laid down that typhoid fever of swine, otherwise called soldier disease or red disease, shall be deemed to be a disease (under the Contagious Diseases Animals Act, 1878) for the purposes of slaughter and compensation, notice of disease, Orders of Council, power of police, and power of entry; and, also (under the Animals Order, 1878), for the purposes of movement and exposure, movement of dung, burial or destruction of carcasses, and general provisions, as also for cleansing and disinfection; but this order has been very imperfectly carried out.

Preventive inoculations have not been tested in this country, but Billings (Nebraska) claims that he has successfully induced immunity by inoculations with attenuated cultures, and Cornil and Chauternes are said to have succeeded (Freidberger and Fröhner) in attenuating the virus by subjecting the cultures to air and heat 93° F. At the end of ninety days the virus no longer kills, but gives immunity to the rabbit and guinea-pig. This attenuation is transmissible to a series of cultures, and with these the following experiments were made. Four pigs were successively inoculated subcutaneously at eight days' interval, with a dose of two cubic centimetres of bouillon cultures, which were ninety, seventy-four, fifty-four, and eight days old, and finally with virulent virus. During the days following the inoculations the animals showed only a slight

illness. Two months after the beginning of the experiments they received in their food one litre of virulent culture in bouillon. Two pigs which were not inoculated were subjected to the same treatment. Within ten days these latter died with characteristic intestinal lesions. The inoculated animals did not show any important morbid phenomenon for two months ; after this lapse two died. The autopsy showed intestinal lesions, which were much developed, and had a chronic course, and very slight pulmonary lesions. Both of the other inoculated animals resisted.

These first experiments showed that the pig acquires immunity with more difficulty than the subjects of other species.

## CHAPTER XXVIII.

### CONTAGIOUS DISEASES—*continued*.

#### SWINE PLAGUE.

SWINE PLAGUE, *rouget de porc*, angina, petechial or spotted fever, red disease, erysipelas, &c., is common on the Continent of Europe, and is, according to Continental writers, a specific septicæmia due to a fine bacillus resembling that of Koch's mouse septicæmia. If inoculated in the mouse, the animal invariably dies; it is also often fatal in the rabbit, guinea-pig, birds, and the pig, but, according to Kitt, the field-mouse possesses immunity, as in septicæmia, to the action of the microbe, and he has also observed that its virulence is attenuated by once passing through the rabbit, and that after six days pigs may be inoculated with blood exudate collected at the point of inoculation, and thus obtain immunity. The microbe exists externally in different media, in damp earth, in water, and in plains and valleys with slow flowing streams. Cornevin states that the horse, ox and sheep, and guinea-pig are immune to its action, as already stated; it is a very fine cylindrical bacillus, measuring from  $\frac{1}{25000}$  to  $\frac{1}{12500}$  in length by  $\frac{1}{42300}$  in breadth; it is found in the blood, particularly in the capillaries in contact with their intima, in the white corpuscles, spleen, liver, kidneys, lymphatic glands, the bone marrow, exudates, fæces, and urine; is non-motile, anaërobic, but grows in contact with air, and in a temperature as low as 45° F. According to Kitt, it resists putrefaction. Schottelius says that it develops spores; but this is denied by Cornevin and Kitt, who base their objections on the non-virulence of dried virus.

This microbe, in reality the discovery of Löffler and Schutz, although ascribed to Pasteur and Thuillier, is destroyed by slow desiccation in from fifty to eighty hours; by water at a tempera-



ture about 83° F. in twenty minutes, and in two minutes at that of 168° F. Cold of about 20° F. kills it in about a fortnight, and salted meat within a month. It is, however, stated that it resists the action of saturated solutions of boracic acid, benzine, chloride of zinc, &c., but is killed by chloride of lime, quicklime, &c.

*Symptoms.*—After a period of incubation of about three days, the disease is manifested by intense fever, constipation, red or purple patches upon the skin, particularly of the abdomen, lower parts of chest, ears, inner aspect of the thighs, &c. There is often grinding of the teeth, muscular twitchings, vomiting, great weakness, paralysis of the hind quarters. The conjunctiva are dark red or reddish-brown in colour, and the patches on the skin become confluent, change from a clear red to dark red, and finally a bluish colour.

The constipation is succeeded by diarrhœa, the fæces being often bloody and containing much mucus, and finally the breathing becomes much accelerated, the surface of the body bluish in colour. Death may occur in twenty-four hours or on the third or fourth day, but is seldom prolonged to over more than a week, and the percentage of deaths is from 50 to 85 per cent., or even higher.

A cure has been attempted by the administration of emetics and calomel, but more attention is now paid to the arrest of the disease by segregation, disinfection, destruction of the carcasses, and finally preventive inoculation has seemingly been of great value in some instances, whilst in others the benefits have been doubtful, and further researches will have to be made before its adoption can become universal. The *post mortem lesions* indicate a more general septic condition; there is congestion of the spleen, liver, kidneys, and endocardium, with hæmorrhage into these organs and into the muscular structures. The spleen externally, however, is tense and resistant from distension of its capsule, but is soft internally and of a reddish-blue colour, and generally unassociated with hæmorrhagic infarctions or extravasations.

There is an intense congestion of the mucous membrane of the stomach and bowels, which is sometimes of a uniform dark red colour, swollen, covered with superficial hæmorrhagic and diphtheritic patches; sometimes it has a marbled appearance. The solitary glands and Peyer's patches are swollen, often covered with bloody mucus, surrounded by a red zone and very often ulcerated, as in the typhoid of man.

## INFECTIOUS PNEUMONIA OF SWINE

is manifested by accelerated breathing, pulmonary gangrene, and caseous tumours, induced by an ovoid *non-motile* bacterium similar to that of the septicæmia of the rabbit and the bacillus of chicken cholera, measuring about  $\frac{1}{200000}$  of an inch in length and about  $\frac{1}{500000}$  in breadth, proving fatal, when inoculated, to the pig in from twenty-four to forty-eight hours. It induces an inflammatory œdema at the point of puncture, and proving fatal in sixty hours in a pig which had been given immunity against swine plague by inoculation. According to Schutz, the contagium is very subtle and induces the disease by inhalation by the skin and digestive apparatus, is rapidly fatal, and is marked by redness, tumefaction of the skin in the regions of the neck and legs; with cough, difficulty of breathing, intense fever, and great depression.

The *post mortem* appearances are those of pneumonia, with several reddish grey hepatised spots, having yellowish necrosed spots in their centres of the size of a grain of sand, and associated with pleuritis and fibrinous pericarditis. The bronchial glands are enlarged, the liver, kidneys, spleen, and heart are softened and degenerate, but the lymphatics of the intestine are not usually altered; and in some chronic cases of the lungs, lymphatic glands, tonsils, bones and tendons, caseous tumours, resembling those of tuberculosis—but not containing the tubercle bacillus—are discovered.

The disease can be inoculated into the rabbit, mouse, guinea-pig, fowls, and pigeons, but large doses are required to induce fatal results in the three latter. It is mostly transmitted from one pig to another by inhalation.

## CHAPTER XXIX.

### CONTAGIOUS DISEASES—*continued.*

#### SWINE ERYSIPELAS.

Much confusion has taken place, owing to the somewhat similar external lesions that are seen in *Swine Fever* and in *Swine Erysipelas*, and also of many other porcine diseases.

It is a well-known fact that pigs, much like infants, if they become sick of almost any disease, are sure to have a skin eruption of some description; hence the difficulty or impossibility of diagnosing disease by the skin lesion alone.

Eggeling distinguishes four forms of swine erysipelas:

1. *Sporadic erysipelas* of the head, due to infection of wounds.
2. *Urticaria*, due to dietetic error.
3. *Epizootic erysipelas*, an acute exanthematous disease similar to scarlet fever in man, and involving both skin and mucous membranes.
4. *Contagious pneumonia* of the pig, a most serious and prevalent form, involving skin, lungs, stomach, and bowels particularly (see p. 334).

Löffler states there are only two forms—namely, swine erysipelas and contagious pneumonia.

The disease is due to a very fine rod-shaped, non-motile bacillus found in the blood, the organs, and the excretions of affected animals, and in Great Britain usually runs a chronic course, and is specially marked by the *post mortem* lesion of *verrucose endocarditis*. During life there may be no symptoms, or, if any, they are vague and often overlooked.

In some countries, however, the disease assumes a virulent form, and causes great mortality; and in these countries resort is had to protective inoculation.

In the acute form the symptoms and *post mortem* appearances are well described in Captain Hayes's translation of Freidberger and Fröhner's "Veterinary Pathology" (Hurst and Blackett), and are as follows :

"*Symptoms*.—After a period of incubation of at least three days, the disease usually begins suddenly and violently. The animal refuses its food ; sometimes vomits or makes efforts to vomit ; has a very high temperature (up to 43° C.) ; manifests severe nervous disturbance ; is very weak, torpid, sleepy, and indifferent to its surroundings, and tries to hide itself under its bedding. The hind-quarters become weak and paralyzed. Muscular spasms and grinding of the teeth are sometimes observed. At first there is constipation ; the mucous membrane of the eyelids is of a dark red or brown-red colour, and the eyelids are sometimes swollen. Usually, a couple of days after the first manifestations of the symptoms, or even from the very commencement of the attack, spots appear on the thin parts of the skin, such as the region of the navel, lower surface of the chest, perineum, inner surface of the thighs, ears, and throat. These spots, which at first are bright red and about the size of a man's hand, become, later on, dark red or purple, and soon unite into large, irregularly shaped patches. As a rule, they are neither painful to the touch nor prominent, but sometimes show a slight inflammatory swelling. The skin of the red spots, especially of the ears, may suffer from an eruption of vesicles, and may even slough. This spotted redness of the skin may be very slight in severe cases, or may appear only immediately before, or even after death. At other times it may be entirely absent, or may become spread over the entire body. Diarrhœa also sets in, and the fæces become thin, mucilaginous, and sanious in a few instances. Towards the end, respiration becomes greatly accelerated, general cyanosis (œdema of the lungs) supervenes, and death takes place, usually on the third or fourth day of the attack, with increased general weakness and considerable fall of temperature (down to 37° C. or less). When the disease is very severe, the animal may die in twenty-four hours. Sometimes the disease takes a week or longer to run its course.

"Endocarditis of erysipelas has been carefully studied by Bang. It often begins acutely with the ordinary symptoms of

erysipelas, and is followed by a latent period, during which the animal apparently recovers ; but subsequently becomes affected with a well-marked, visible, cardiac affection, which generally lasts for a week or two. In a few cases the pigs die from apoplexy. The symptoms are: loss of appetite, depression, persistent lying down, shortness of breath, slight cough, and redness of the skin, in varying intensity and extent ; although not to such a high degree as in acute erysipelas. After this, we find palpitation of the heart, greatly accelerated action of the organ, and sometimes endocardial murmurs. The temperature is often increased and the animal dies with symptoms of cardiac paralysis. In a few cases we may observe paralysis of the hind-quarters. On *post mortem* examination the left heart is usually found to be affected ; the right, less frequently.

“ *Prognosis.*—The ordinary kind of epizootic erysipelas has a mortality of from 50 to 85 per cent. Lydtin states that the losses from it in Baden were between 50 and 75 per cent.

“ *Anatomical Changes.*—In *post mortem* examinations of cases of the ordinary form of epizootic erysipelas, we find signs of septicæmia without any well-marked morbid conditions of separate organs. This septicæmia is a general infection which produces hæmorrhagic and diphtheritic gastro-enteritis, considerable swelling of the lymphatic system, hæmorrhagic or parenchymatous nephritis, acute swelling of the spleen, parenchymatous hepatitis, and myositis.

“ 1. The hæmorrhagic gastro-enteritis consists at first of excessive inflammation of the mucous membrane of the stomach in the region of the fundus. The mucous membrane shows a dark red discoloration, which is partly diffuse, partly in spots ; suffers from cloudy swelling ; often has eminences on its surface ; is covered with a viscid layer of mucus ; and may even have superficial scabs. The glands of the mucous membrane are inflamed (gastritis glandularis). The intestinal mucous membrane is swollen, especially on the top of the folds, narrow parts of the small intestine, and in the neighbourhood of Peyer’s patches ; is covered with reddish mucus ; infiltrated with hæmorrhages ; and sometimes shows superficial scabs. Less frequently, circumscribed parts of the mucous membrane of the cæcum and of the anterior part of the colon suffer from a diphtheritic affection.

"2. The solitary follicles and Peyer's patches present, throughout, medullary swellings in the form of prominent raised patches of the size of a grain of millet to that of a lentil. Sometimes they are infiltrated with hæmorrhages, and surrounded by a red ring. We very frequently notice ulceration and cicatrisation of the solitary and agminated follicles. The mesenteric glands become more swollen than the other glands of the body, are of a dark red colour, and show softening. The surface of their section is dun-coloured, with interspersed dark red spots, and the paraglandular tissue is hyperæmic and infiltrated with hæmorrhages.

"3. The hæmorrhagic nephritis is distinguished by enlargement of the kidneys, and by the kidneys assuming a grey-red colour. The medullary layer is generally of a very dark red, and the cortical layer is infiltrated with blood points, and widened out. In slight cases we find only a parenchymatous inflammation (cloudy swelling) of the kidneys. Frequently catarrhal nephritis occurs as a complication.

"4. Acute swelling of the spleen arises in consequence of an acute, severe hyperæmia, with great increase of the cellular constituents of the spleen (new formation of splenic cells), in which case the spleen is enlarged, but not softened as in anthrax. It is, on the contrary, tense to the touch, as the capsule is considerably stretched. The pulp on the surface of a section is purple, moderately soft, and free from hæmorrhages.

"5. The parenchymatous hepatitis consists of a cloudy swelling and enlargement of the liver, in which the surface of sections has a greyish-brown colour, soft, flaccid, watery, shiny, are sometimes infiltrated with hæmorrhages, and look as if they had been boiled. The myocardium shows similar spotted changes and subendocardial hæmorrhages.

"In the abdominal cavity, thoracic cavity, and pericardium we sometimes find small quantities of an orange-coloured clear fluid, which may be mixed with flaky coagula, and punctiform hæmorrhages under the serous membranes, especially on the auricles. We rarely meet with severe hæmorrhages in the brain, or in the dorsal and lumbar portions of the spinal cord. The congested parts of the skin are somewhat discoloured after death. The lungs remain un-

changed, or at most exhibit a *post mortem* œdema. By microscopical examination, the bacilli are found everywhere in the body, especially in the spleen and kidneys, but to a less extent in the blood."

In our experience the patches on the skin are frequently diamond-shaped, and contain the specific organisms.

In some cases large patches of the skin slough off, and leave large ulcerating surfaces.

Mr. Blackhurst, of Preston, has seen numbers of people associated with such diseased pigs affected with erysipelas of the face.

There are three recognised methods of preventive inoculation :

1. *Lorenz's Serum Method*.—The simultaneous injection of immune serum and virus at different parts of the body.

2. *Leclainche's Method*.—Inoculation with a serum virus mixture, followed in twelve days by the injection of a pure culture of the organism without any serum.

3. *Pasteur's Method*.—Inoculation with two vaccines at different degrees of virulence. This is done by passing the virus through rabbits, whereby it gradually loses its virulence, until it no longer kills swine.

This attenuation of virulence is maintained if the virus be grown in bouillon.

Pasteur uses two vaccines, an extremely weak one, and followed up in ten days by a stronger.

The procedure is to inject inside the thigh of young pigs 0.1 c.c., and immunity is thus obtained in a fortnight and maintained for a year.

## CHAPTER XXX.

### CONTAGIOUS DISEASES—*continued.*

#### MALIGNANT ŒDEMA.

THIS disease affects all classes of animals, and is due to the inoculation of a wound, however slight, with the organism known as the bacillus of œdema of Koch (*Vitrio septicus* of Pasteur).

These organisms are found almost everywhere in the soil—in dust, in fæces, &c., &c.

They are particularly harmful when they obtain a footing in the connective tissue, and especially so when that tissue is in a condition of irritation or inflammation.

They are anaërobic and motile, and are destroyed by oxygen; but their spores resist ordinary disinfectants. When the bacilli gain entrance to a wound, and when there is little or no blood and no oxygen, they grow and multiply by division, and by their action cause the evolution of several gases—namely, sulphuretted and carburetted hydrogen, hydrogen, and carbonic acid—which gases accumulate under the skin, and give rise to emphysema.

Invasion of the wound by the bacilli results in a febrile condition, a gradual loss of sensibility, and ultimately coma. The swelling extends rapidly, and the affected parts become of a bluish colour, a leaden hue prevails, and finally the part becomes cold and gangrenous.

Should the wound which becomes infected be in the neighbourhood of the throat, the swelling will so much interfere with respiration as to cause suffocation.

The disease is frequently seen in lambs after castration and tail-cutting, and terminates fatally in a gangrene of the abdominal muscles or of the hind-quarters.



The bacillus of oedema is frequently found in the blood of *dead* animals, and has been mistaken for the *Bacillus anthracis*.

The treatment for the disease is to freely incise the swelling, so as to admit oxygen, and to irrigate the part with antiseptics.

When the throat is swollen, it may be necessary, in addition to the above treatment, to perform tracheotomy.

*Post mortem* reveals, in addition to the local swelling, which, when cut into, is found to contain large quantities of orange-coloured frothy exudate, an exudation of serum into the substance of the lungs, and very often an inflamed condition of the bowels.

The spleen is found to be normal.

## CHAPTER XXXI.

### CONTAGIOUS DISEASES—*continued.*

#### SOUTH AFRICAN HORSE-SICKNESS.

THIS is one of the most fatal of diseases affecting the horse in South Africa, and is said by Duncan Hutcheon to be due to the inoculation of an ultra-microscopic organism, through the agency of a nocturnal insect, and the disease can be transmitted to other horses by injection of the blood of diseased animals.

The period of incubation is said to be about eight days, but no special indications of illness may be noticed until an hour or two before death, and in many cases a horse is found dead which had seemingly been in good health a few hours before.

The symptoms that have been recognised are great depression, elevation of temperature, hurried respiration, a well-marked swelling of the pits or hollows above the eyes, and the visible mucous membranes become of a dark red hue.

On auscultation of the lower part of the trachea and the larger bronchial tubes, a bubbling sound is heard, due to effusion. This effusion is great, and as the inspired air mixes with it, it is discharged from the nostrils as a copious yellow froth, and in a very short time the effusion into the tubes and vesicles is so great that the animal dies of suffocation.

On *post mortem* examination one finds great effusion into the lung substance, the bronchi and vesicles, the pericardium, and the pleural cavity. Effusions are also seen subcutaneously in the lower cervical region. The blood is black, but coagulates quickly. The line of procedure is purely defensive, and is to keep the animals in a stable, if possible, or in a kraal, and seemingly this *prevents infection*.

## CHAPTER XXXII.

### CONTAGIOUS DISEASES—*continued.*

#### SPIRRILOSIS IN THE HORSE.

THIS disease is due to the presence of spirilla in the blood, and is characterised by general depression, emaciation, and œdematous swellings of the upper eyelids (like horse-sickness), of the neck, and of the dependent parts of the body.

The disease seems to run a rapid course; the emaciation is great, accompanied by much debility and loss of appetite, terminating in death.

An examination of blood-smears usually shows the presence of spirilla.

On *post mortem* examination the swellings are found to consist of a yellow gelatinous exudate, and the kidneys to be pale and enormously enlarged (as described by R. T. Sturdy, M.R.C.V.S., they may weigh 56 ounces each even in a pony), and the bladder to be filled with pale urine.

The muscular tissue is noticed to be in a seeming parboiled condition, and the *rigor mortis* is very slight.

The most noticeable appearance is the great emaciation, Mr. Sturdy's case wasting from a well-conditioned pony to a mere bag of bones in five days.

## CHAPTER XXXIII.

### CONTAGIOUS DISEASES—*continued*.

#### SPIROCHÆTOSIS.

THIS is a specific blood disease, due to the presence of small, thin, and very active parasites, which have the appearance of young and thin eels, writhing and wriggling with great rapidity.

They have also been found by Jowett in the discharges from cases of "grease" and "canker" in the horse.

The parasite was first demonstrated by Obermeier in the blood of a patient suffering from relapsing fever.

These parasites have since been demonstrated in horses, cattle, sheep, pigs, and birds, and are known as—

*S. Duttoni* : The cause of tick fever in cattle in Africa ; also supposed by some to be the cause of red or black water in cattle.

*S. Theileri* : A similar parasite to the above, and described by Theiler as occurring in African cattle in 1902.

*S. ovina* : In sheep.

*S. suis* : In pigs.

*S. anserina* : In geese.

*S. gallinarum* : In birds.

*S. Obermeirei* : In man. The first demonstrated.

*S. pallida* : In syphilis of man.

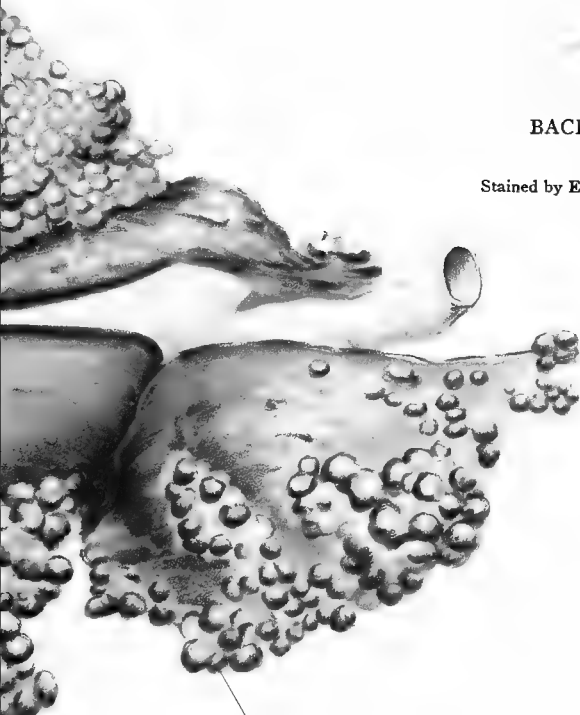
*S. of Jowett* : In "grease" and canker of the horse.

*S. suis* gives rise to progressive anæmia in pigs, and has a period of incubation of about fourteen days ; is characterised at first by the appearance of certain cutaneous lesions—namely, firstly, a desquamation of the epidermis, in small, circular patches ; then an extravasation of blood, which dries up, leaving a brown scab ; and later on the scab drops off,





TUBERCULOSIS OF OU



BACILLUS TUBERCULOSIS.

Stained by Ehrlich's method from specimen in College  
Laboratory. X 2000.

Tubercles on surface  
of Lungs.

OF LUNG. ("GRAPES.")





and leaves a permanent cicatrix, rather like a pock mark, and in white pigs, intensely white, and glistening like pearl. The spirochætæ were always found in the primary lesion, but never in the blood, and they are not found on or in the skins of healthy pigs. The disease is highly contagious, and frequently terminates in death from anæmia, even though the acute skin lesions have disappeared. (Sydney Dodd, M.R.C.V.S., is the first to demonstrate this disease.)

## CHAPTER XXXIV:

### CONTAGIOUS DISEASES—*continued*.

#### COCCIDIOSIS.

Coccidia are protozoa, which assume the form of ovoid cysts, varying in length from 18 to 25  $\mu$ , and in width are about 13  $\mu$ . They possess a hyaline envelope whose existence is proved by its double contour and yellowish, granular, highly refractile contents. This granular material does not always occupy the whole of the cavity. At a certain stage of development it collects towards the centre, forming a nucleus, and leaving clear spaces at the poles of the cells. At a later stage this nucleus divides into four portions, which afterwards separate. These protozoa live in wet, marshy places, but are destroyed by drying in the open air (Degoix), and are the cause of fatal diseases in cattle, hares, rabbits, and fowls.

In cattle the disease is often called bloody flux, hæmorrhagic enteritis, and dysentery.

After infection, it takes about six weeks to two months before the coccidia have become sufficiently numerous to cause disease.

It affects cattle of about eighteen months to two years of age, and usually breaks out in midsummer.

The first symptom is a serous, foetid, dark greenish diarrhœa, this fluid being voided without any straining or pain. Within a few days, however, the animal is seen to stand with arched back, to show signs of abdominal pain, and to grind its teeth. The character of the ejeeta is much altered. It is now mucous, sanguinolent, and often contains blood-clots. This causes considerable pain, tenesmus, and violent straining. The fluid is expelled in large quantities.

Signs of fever appear; the temperature rises; the animal ceases to feed and to ruminate; great thirst is exhibited; rapid emaciation takes place; the animal becomes very weak and hardly able to stand. Then a change occurs in the course of a few days—a week to ten days. Either the animal is so weakened that it dies, or if any appetite had been present during the acute stage, the symptoms gradually disappear, and recovery takes place.

In some cases, particularly in animals in good condition, after the initial stage is passed, a bloody flux has set in, the animal has violent convulsions, lies on its side, head stretched out, legs stiff and straight, eyes retracted, and sometimes there is opisthotonos. The convulsions occur time after time, and terminate in death.

Treatment consists of the administration of alkaline, mucilaginous drinks, and of weak solution of creolin or salol, accompanied by easily digested and rich foods, followed by a course of vegetable tonics (Degoix).

As the disease is contracted by the animals pasturing on infected lands, all those not affected should be removed, and preventive treatment adopted.

All ejecta from infected animals should be sprayed or dressed with a 3 per cent. solution of sulphuric acid.

*Post mortem* examination reveals the fact that the large intestines have been the seat of the disease. They are found to be almost entirely empty, the mucous membrane to be oedematous, reddish-brown in colour, and covered with mucus, sometimes having the appearance of grey, diphtheritic patches, and which on removal leave a whitish ulcer, and it is principally in these patches that the coccidia are found.

Coccidia are found here in greatest number, though they are seen almost as numerous in the epithelial cells of the follicles, and as often they are found in masses at the bottom of the much-dilated blind ends of the follicles, and contained with the epithelial cells, which have undergone considerable hypertrophy.

## CHAPTER XXXV.

### CONTAGIOUS DISEASES—*continued.*

#### TUBERCULOSIS.

*Definition.*—An infectious, infective, and inoculable disease induced by the action, particularly upon the hereditarily predisposed, of a bacillus discovered by Koch in 1882, and termed the *Bacillus tuberculosis*, affecting many kinds of animals, and in consequence called by Lydtin the “universal panzootic.”

Tuberculosis is most commonly seen in man, monkeys, and horned cattle, less frequently in the pig, horse, dog, and cat, and birds; amongst the latter, however, it sometimes assumes an epizootic form, attacking poultry, pigeons, pea and guinea fowls, turkeys; even small birds not being proof against its ravages. The smaller ruminants, sheep and goat, are very rarely attacked with tuberculosis; they may resist subcutaneous inoculation, but they are not proof against its intravenous inoculation. The direct introduction of the bacilli into the blood, and the repeated ingestion of tuberculous growths, induces the disease even in them. It is also found in the camel, giraffe, antelope, llama, gazelle, zebra, &c., and it is now well known that the disease is transmissible from man to animals, and from animals to man.

The tubercular nodules vary in appearance, some being grey, like pearls, hence the term “pearl disease” in England, “*perlsucht*” in Germany; some being yellow, and some calcareous, these varying appearances indicating the age of the growths. They are non-vascular, and vary much in size, some being even smaller than a millet seed (miliary tubercle), very numerous, and invading one or many organs. Some larger ones are hard, greyish-red, pedunculated, or having broad bases, arranged in clusters of various sizes, covering the lungs, thoracic walls, surface of diaphragm, the peritoneum, &c.; or they are found in the form of great fluctuating masses filled with yellow pus, which is thick, grumous, full of calcareous grains, resembling mortar, or of an

opaque yellowish matter infiltrating the tissues. These varying appearances gave rise to the opinion amongst Virchow and his followers that they were lesions of different diseases; and Villemin, who first discovered the inoculability of tuberculosis in 1865, vainly tried to prove their identity by inoculations, but it remained for Koch to prove that they were all due to tubercular bacilli.

Examined microscopically, the nodules present giant cells with branched processes and large epithelioid cells, which are round or oval in shape, and outside a zone of lymphoid cells. The cells are surrounded by a more or less complete fibrous stroma. These tubercles are sometimes scattered throughout the substance of the organs, having the form of distinctly rounded nodules, rather gelatinous in structure, and a pearly grey transparent appearance. In other cases it will be found that beneath the pleuræ, both visceral and parietal, there are large yellow nodules, which give off numerous radiating branches. These are called "grapes," from the supposed similarity to that fruit, and between them are seen some red lines in which degeneration has not advanced to any extent. Each yellow patch is seen under a low power to consist of a number of follicles, each being degenerated—caseated—in its centre, embracing the alveoli and their contents. There are no blood-vessels in the centre, but some vessels are seen with difficulty some distance from it, where the alveolar walls are slightly thickened. In order to differentiate the giant cells from those of an epithelial type and from the fibrous capsule, stain with picro-carmin, when the giant cells will present the yellow picric acid colour, and the others will be stained carmine; for the recognition of the bacilli other methods must be resorted to.

There is a difficulty in finding the bacilli in caseous tubercles, particularly in cattle, but they can often be demonstrated in material taken from the periphery of the nodule. If cavities containing pus are formed, such as are not uncommon in tuberculous horses, the bacilli can be detected very readily by the following process:—

A drop of water is placed on a perfectly clean cover-glass. Remove a small portion of the pus or caseating matter with the point of a needle, then rub it with a drop of water already placed on the cover-glass. The cover-glass is then dried in

the air and fixed over a spirit lamp or Bunsen burner until the albumen is coagulated. A few drops of filtered carbol-fuchsin solution is put on the cover-glass, and held by a pair of forceps over the flame until steam rises for five minutes. Wash in water, and decolorise by 25 per cent. sulphuric acid solution, and then wash in water. Counter-stain with aqueous solution of methyl blue, then wash with water, dry over a flame, and mount in Canada balsam. The bacilli will present a bright red colour on a pale blue ground, and occur singly, in pairs, or aggregated in rosettes.

The bacilli vary in number in different lesions, and have no relation to the magnitude of the growths. Thus in a large nodule very few bacilli will be found after the most careful examination, whilst a smaller one may contain a large number. When the giant cells are numerous the bacilli are generally few in number, many of them having doubtless been ingested by the giant cells. They are easily detected in the expectorations of human consumptives, and in the thick purulent yellow discharge which is sometimes ejected from the mouths of cattle after long and violent fits of coughing. This material coughed into the mangers and upon partitions and walls of the cow-shed is, when dry, diffused through the air inspired by the healthy cattle, and thus becomes one of the great causes of the spread of the disease.

The transmissibility by inhalation, as well as that by ingestion and inoculation, has been proved experimentally. The animals experimented upon were compelled for several hours daily, to breathe, in a chamber, air in which were fine particles of phthisical expectoration, mixed with water, from persons with cavities in their lungs, and rendered into fine particles by a steam atomiser. Dogs alone were used, as they rarely suffer from tubercle. Eleven animals were experimented upon, and all were killed in a period varying from twenty-five to forty-five days, and, with one doubtful exception, presented well-developed miliary tubercles in both lungs, and in most of them tubercles were also found, but to a smaller extent, in the kidneys, and to a still smaller extent in the liver and spleen. Microscopical examination demonstrated the presence of the bacilli.

These experiments were conducted by Dr. Tappeinier of Meran, and a preliminary account of them led Dr. Max Schottelius to make similar ones, not only with the sputum of

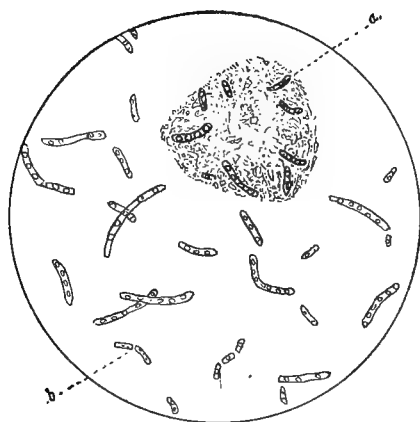


FIG. 30.—Tubercle bacillus.

- a.* Giant cell invaded by bacilli.  
*b.* Minute germs.



FIG. 31.—Tubercular nodule.

- a.* Caseated centre of nodule.  
*b.* Giant cells.  
*c.* Reticulum of nodule with epithelium-like cells.  
*d.* Leucocytes, red cells, &c.  
*e.* Portions of adjacent alveoli.

phthisical individuals, but also with that of persons suffering from simple bronchitis, and with pulverised cheese, brain, and cinnabar. The result was that miliary tubercles were found in the lung in all cases, and in equal quantity with both phthisical and bronchial sputum. Cheese produced a smaller quantity, pulverised brain still less, and the cinnabar least of all, merely a few whitish tubercles with pigmented centres, with an interstitial deposit of the substance, which had caused no inflammatory reaction; though the tubercles produced by non-specific matter closely resembled those of tuberculosis, they contained no bacilli, hence they cannot be classified as those of true tuberculosis.

Tuberculosis is described under three divisions, namely,—1st, Tuberculosis of the organs; 2d, of the serous membranes; and 3d, of the lymphatic glands; but generally these forms co-exist in the same subject, constituting the so-called “diffuse tuberculosis.”

According to Nocard, it is shown that 40 per cent. of tuberculous animals are affected in both lungs and pleura, 20 to 25 per cent. in lungs alone, 15 to 20 per cent. in pleura and peritoneum, the remainder comprising either generalised lesions of all the organs, or lesions localised in the glands, the genital organs, mammæ, tongue, osseous tissue, &c., and that the lymphatic glands proceeding from the organs attacked are always more or less severely affected; and he makes this important statement, that in many cases when the animal is slaughtered quite at the commencement of the disease tuberculous nodules are only found in the bronchial or mediastinal glands; and the pulmonary lesion which has served as the entrance gate for the contagion eludes the most careful research, or is only represented by a minute focus of disease much less important than the glandular alterations which it has preceded and caused.

Bollinger thinks it possible, or rather probable, that intestinal tuberculosis, consecutive to pulmonary phthisis, may be produced by the discharges (sputa) being swallowed and passing into the intestines, mesenteric glands, and liver, without there being generalisation through the blood-stream.

It has now been definitely proved by McFadyean and others that ingestion is a common means of entry for the organism, and that the infection of the lung is secondary to the intestinal infection. The path of contagion may be by the lymphatics, or be carried by leucocytes through the blood.



This has been one of the arduous duties of His Majesty King Edward's Royal Commission, and by its demonstration it has been enabled to show the communicability of bovine tubercle to man. It has been proved that human tubercle, when fed to bovines, will reproduce the disease, but its virulence is somewhat modified. The cultural characteristics of the two are very similar, but the human, after its passage through the bovine, becomes seemingly identical, thus demonstrating that the two are of the same species, but varying slightly under the different conditions. The disease *tabes mesenterica* is therefore undoubtedly produced in children, in numerous cases, by the feeding with tuberculous cow's milk. This is a direct intestinal infection, and not through the medium of the lungs. The bacillus has a natural power of selection, and prefers the lung as a seat of growth; but no matter in what way infection is obtained, there is a probability of early pulmonary infection.

*Symptoms in Cattle.*—Tuberculosis is generally slow in developing, and its symptoms are often very obscure, whilst in a few instances its manifestation may be very rapid. In the latter case signs of ill-health appear, excited, perhaps, by parturition, a slight cold, indigestion with recurrent tympanites, or some other, perhaps trivial, cause; the illness continuing longer than usual, and the animal, being fat, is slaughtered to prevent loss, and the *post mortem* reveals the presence of tubercular tumours in various parts of the economy. In this case some of the tumours will be found in a softened, semi-fluid condition, the contents of which being absorbed, induce the continuance or aggravation of the otherwise trivial illness. Generalised tuberculosis only occurs when the blood-stream has become infected by the entrance of the bacilli. Nocard says:—"It often happens that apparently healthy animals slaughtered for human consumption are found to have several organs invaded by the disease, yet in such condition it cannot be said that the disease is generalised: these are successive localisations, and it is maintained that general tuberculosis only occurs when all the glands in the chain are destroyed, the lymph carries the bacilli into the thoracic duct, whence they are poured into the anterior cava, or, again, when a tubercular focus penetrates a vein of a certain size and pours into it the virulent material which it contains. Tuberculosis then assumes the character of a general disease, and all

the vascular tissues are virulent, and the tissues which are favourable to the growth of the bacilli, notably the liver and spleen and the marrow of the bones, become the seat of a number of specific granulations all of the same size and age, which constitute what is known in human medicine as granular or acute miliary tuberculosis." Miliary tubercle in the spleen is said by Ostertag to be the surest sign of general tuberculosis. Although the animal may be fairly fat, the flesh usually is pale, watery, pitting under pressure, and prone to rapid decomposition.

In deep milkers, and in some highly bred cattle, the cachexia may seem to precede and accompany the tuberculosis, and generally the earliest signs are those of unthriftiness, and deterioration in the quality of the milk, which becomes thin and watery, although for some time it may keep up in quantity. If the cow be in calf, abortion is apt to occur; if not pregnant, the condition called nymphomania is frequently present. The appetite is capricious; the mucous membranes pale; a cough of a dull character exists; the skin looks dull, the hair dirty; the animal does not lick itself, and, in the white parts, the skin is often observed to be yellow. Emaciation now proceeds more or less rapidly; the cough becomes troublesome, but there is seldom any expectoration or discharge from the lungs. The digestive organs are weak, the rumen prone to tympanites, and diarrhoea sets in, which soon renders the animal a mere bag of bones. Auscultation and percussion may find the lungs and contents of the thorax diseased or otherwise. There is generally some degree of pain and tenderness evinced by the animal when the sides are sharply struck or pressed upon, and very often a friction (pleural) sound is heard, and pressure upon the loins will cause the animal to cringe, groan, or otherwise evince pain.

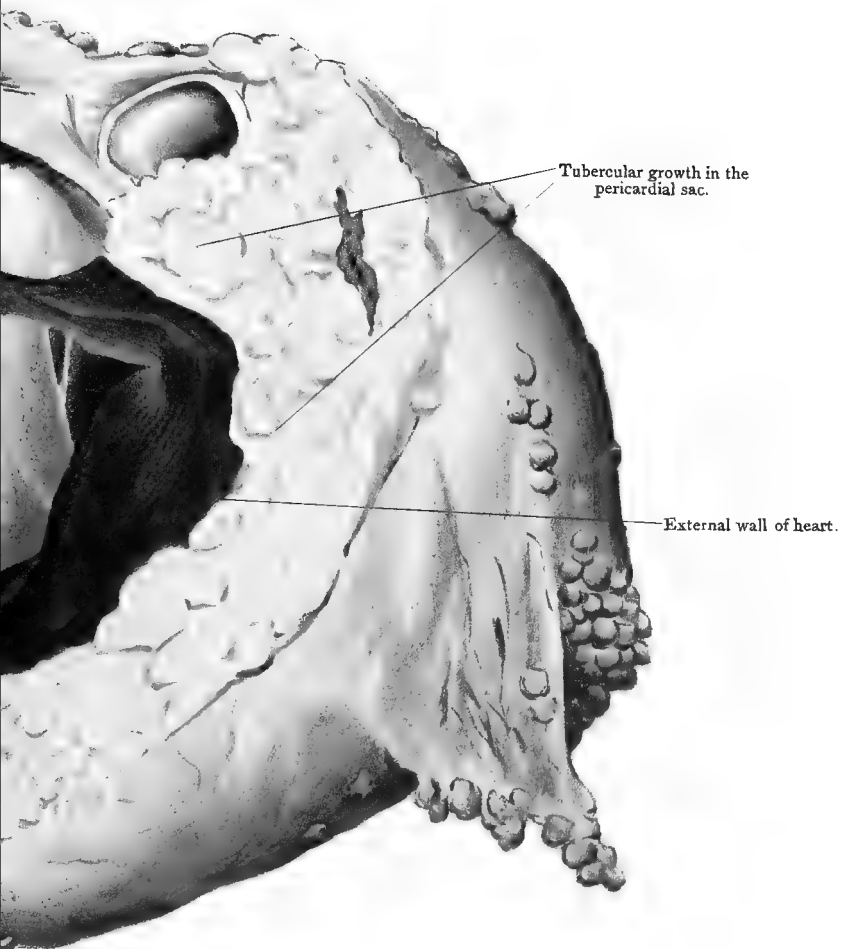
As the disease advances the cough becomes more troublesome,—paroxysmal; louder than that of pleuro-pneumonia, and occasionally bronchial discharges may issue from the mouth after a severe fit of coughing. As already stated, in this yellow, grumous, and viscid material the bacilli may be detected by microscopic examination. It, however, most frequently happens that the severest fit of coughing brings about no discharge, the material being swallowed. This also occurs in pulmonary glanders in the horse, in which abscesses may be found on *post mortem* examination filled with pus, freely communicating with





The Heart and Lungs were

TUBERCUL



v, and together weighed 56 lbs.

ERICARDITIS.



the bronchi, from which during life no discharge has reached the nostrils, having been swallowed, as may be observed by watching the animal. It can be seen by standing at the left side, that the act of swallowing is performed, and the course of the material followed by the eye during its course from the pharynx into the chest.

The animal rapidly becomes more or less hidebound, the hair dull, the expression dejected, the eyes watery and sunken, and the lids often covered with a scaly material, whilst a foetid discharge may issue from the nostrils. The respirations, as the disease advances, become greatly accelerated, short and jerky, each expiration being often associated with a moaning or grunting sound; the prostration is extreme, and the animal soon succumbs.

On percussing the walls of the chest the animal evinces some signs of pain, and a cough is often excited; there is decreased resonance and often signs of lung consolidations and of pleural change, whilst auscultation may reveal increased tubular sounds with diminished or absence of the vesicular murmurs, and often signs of consolidation. There may also be pronounced bronchial rales when the discharges have invaded the bronchi, and friction sounds when the pleural surfaces are roughened by the sub-pleural nodules and adhesions of the pleural surfaces. Some authors state that the external lymphatic glands are frequently enlarged: so far as my experience goes this is exceptional, and of no great diagnostic value. The temperature varies considerably in individual cases; in some very pronounced ones there is no great elevation, whilst in others this is extreme,— $107^{\circ}$  or higher during the exacerbations, falling several degrees during the remissions, and these exacerbations and remissions are not infrequently met with in the pulmonary form.

As already stated, the calf is rarely tuberculous, and from the observations made at the abattoirs at Berlin and Copenhagen it was found that at Berlin the percentage of tuberculous animals was 15.1 for oxen and cows, 1.55 for swine, 0.11 for calves, and 0.004 for sheep. At Copenhagen the percentage was 17.7 for oxen and cows, 15.3 for swine, 0.2 for calves, and only 0.0003 for sheep. From its rarity in calves some writers have argued that tuberculosis is not congenital. This conclusion, however, is against evidence, for cases are recorded where

undoubtedly the calf has given evidence of the disease at or shortly after birth, where the lungs and various serous membranes—*i.e.*, pleura and peritoneum—were covered with caseous and calcareous tumours, which must have been developed *in utero*. In two instances which came before me the arachnoid was covered with tubercles in an early stage of development, the calves dying from meningitis in an acute form. Infection of the foetus results from the penetration of the bacilli through the foetal membranes, or from their presence in the semen of the male or ovum of the female at the time of coition. We can understand this when we recognise that the ovaries of the female and the testes of the male are occasionally the seats of tuberculosis.

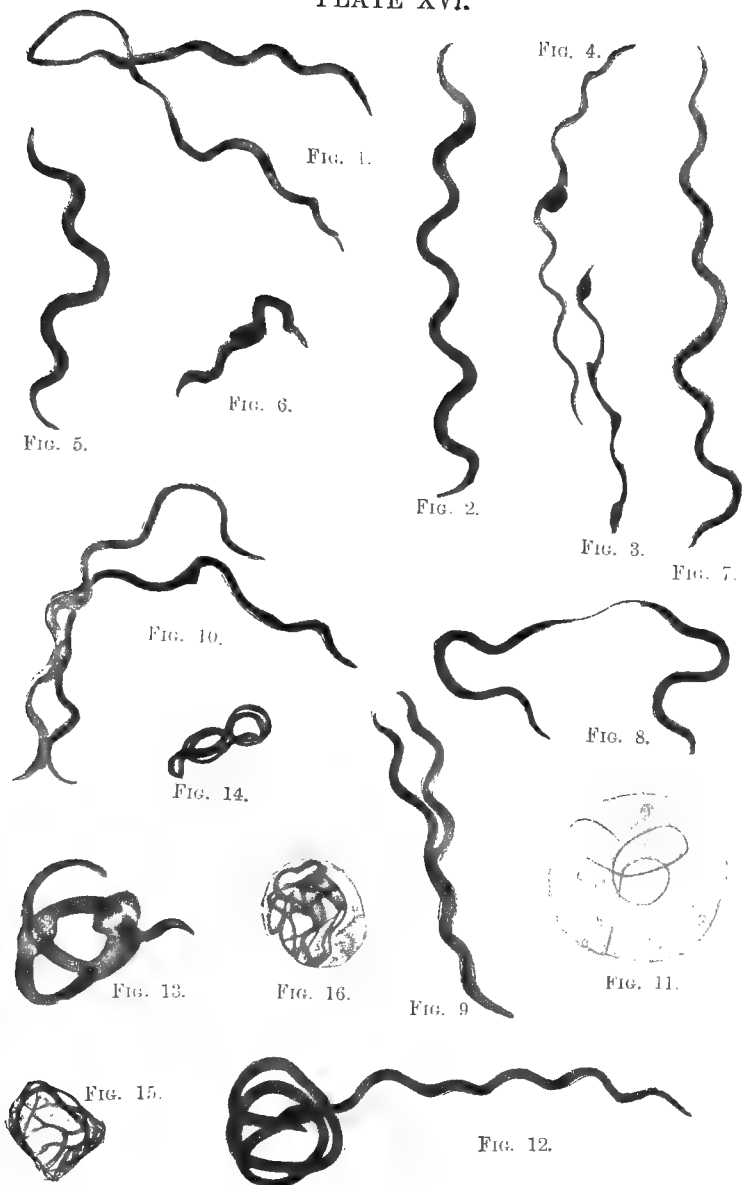
The liver, spleen, heart, mesenteric glands, and peritoneum may be invaded with many clusters of tubercles similar to those shown in figs. 15 and 16, whilst not infrequently the ovaries and uterus are involved, causing continuous sexual excitement,—nymphomania,—and sometimes discharge from the vagina, with non-impregnation after repeated serving; but should pregnancy occur, the animals generally abort, and according to Roloff this abortion is often the first sign of the infection of a herd.

Tubercle is also found invading the meninges of the brain and spinal cord, giving rise to fits of convulsions; to ptyalism and dysphagia and tympany when located in the pharyngeal glands; to purulent discharge when involving the penis of the male, uterus or vagina of the female; and colic, with alternating constipation and diarrhoea, and flatulency after food, particularly if the liver be involved. The pericardium may be considerably affected without apparent symptoms, but after a time signs of cardiac irregularities become more and more apparent (see Heart Diseases), and sometimes the external lymphatic glands are enlarged; whilst in other cases an animal apparently quite healthy manifests symptoms of lameness from no assignable cause, and this lameness is generally irremovable, resisting all treatment, and after death the bones or joints present the usual characteristics of tubercular disease.

Tuberculosis, when affecting the mammary gland, is very frequently secondary to its development in other organs, but is supposed to be occasionally primary. Bang, Copenhagen,



# PLATE XVI.



SPIROCHAETA DUTTONI FROM MONKEYS AND RATS.  $\times 4,500$ .

FIGS. 1 TO 12: From peripheral circulation of infected monkeys and rats.

FIG. 13: From monkey's liver.

FIGS. 14 TO 16: As found in the spleen.

From the Report by Dr. Anton Breinl, Director of the Runcorn Research Laboratories of the Liverpool School of Tropical Medicine, and published in the Annals of that School, vol. i., No. 3.



describes the condition as a diffuse, painless, hard swelling, involving one or more quarters of the gland, but generally the posterior ones. It frequently is secondary to an ordinary mammitis, and after the acute symptoms have subsided the quarter slowly but continuously increases in size. It differs from an ordinary inflammation of the mammary gland—*mammitis* or *mastitis*—in which the milk is curdled at commencement; but in the tubercular form the milk is at first normal, but after a time it becomes watery, contains clots, and sometimes bacilli.

Bacilli are found with great difficulty; and Bollinger states that in fifty-five per cent. of examined cows where the milk was virulent the bacilli could only be found once in twenty cases. There is therefore great danger to human life when the milk of tuberculous cows is made use of as food. No doubt innumerable cases of consumption are thus induced in the human being, particularly that form of tubercular disease *tubes mesenterica*, so common in young children.

It must be particularly borne in mind by the veterinarian that, as a conservator of public health, he should at all times discountenance the consumption of the unboiled milk of tuberculous cattle not only by human beings, but by the lower animals, for it has been abundantly proved that the milk of a tuberculous cow will, unless boiled, be dangerous to life; and when we reflect upon the fact that it is so largely consumed, particularly by infants and young children, we can at least imagine the appalling consequences it may give rise to without being boiled.

The danger to human life from the ingestion of the milk of a tuberculous cow is greatly increased if its mammary gland be in a condition of inflammation, the seat of an abscess, or in a state of induration, as—in addition to the deficient nutritive value of all milk from tuberculous cows, which is blue in colour, thin in consistency, and in some cases contains cyanides—it will in such an instance be tainted with bacilli, and be a direct means of infecting the human being, particularly young and delicate infants. The boiling of milk and the thorough cooking of meat destroys with certainty the bacilli, and all suspected milk should be so treated. Let the veterinary surgeon, when called upon to give his opinion, consider the

sacredness of his position, and—even if he chance to offend a client—do his duty towards his fellow-beings.

The majority of the veterinary profession are in favour of preventing the flesh of all tuberculous cattle—no matter what its condition and appearance may be—being used as human food; but I cannot support the wholesale condemnation of such flesh; and, whilst maintaining that such flesh, when properly dressed, and all tubercular growths with adherent flesh have been carefully removed, is fit for food, I must explain that if it present any appearance of being watery, pale, or otherwise unhealthy, I should not hesitate in at once expressing my conviction that it should not be used as food for man.

The following abstracts from the *Report of the Royal Commission*, appointed to inquire into the administrative proceeding for controlling danger to man through the use as food of the meat and milk of tuberculous animals (Part I., 1898), will be found of much interest and value:

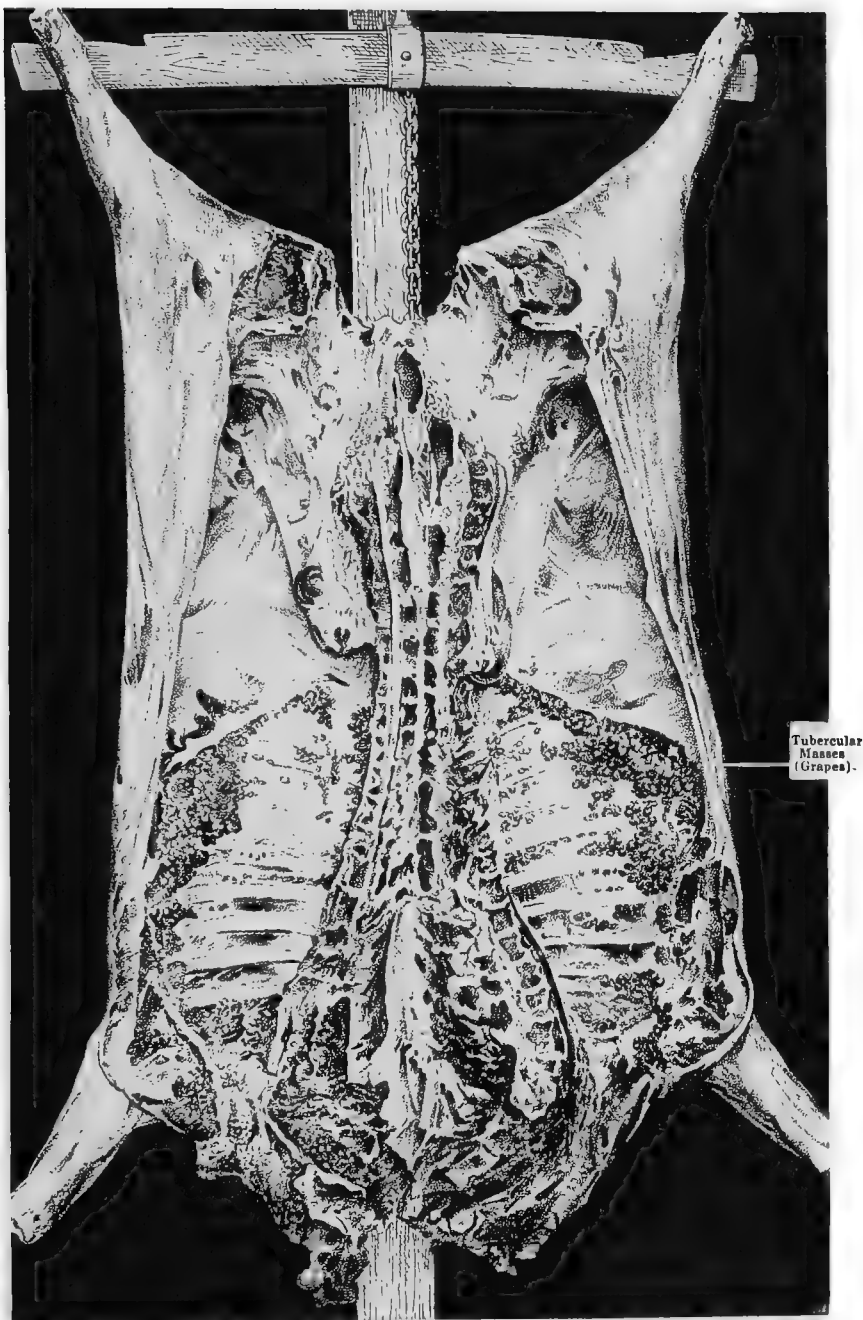
There is a total absence of uniformity in the special qualifications required of the persons employed as meat inspectors by the sanitary authorities in different places, as may be seen by a Return presented to the House of Commons in 1896, showing the previous vocations of those acting in that capacity. In Battersea, for instance, four plumbers and three carpenters discharge the office of meat inspector; in Hackney the duties have been committed to two plumbers, one carpenter, one compositor, one bricklayer, one florist, one builder, one surveyor, and one stonemason. In Portsmouth a solitary butcher has received as colleagues three school teachers, one medical dispenser, one carpenter, and one tram-conductor.

In Manchester the public slaughter-houses are under supervision of a veterinary surgeon; in Glasgow inspection is carried out by the police, assisted by three butchers, and in Liverpool the staff of inspectors have been “butchers by trade and training.”

We may add that in the Edinburgh public slaughter-house we witnessed meat inspection carried on more nearly on the enlightened system of the best Continental abattoirs than it was our fortune to see in any other part of the United Kingdom. Here there are six meat inspectors, of whom four are veterinary surgeons, one has been a butcher, and one a cattle salesman. We were very favourably impressed with the organisation, though the standard by which the meat of tuberculous carcasses was judged appeared to us unnecessarily severe.

In many districts meat inspection is made part of the duty of ordinary sanitary inspectors, without any special training.

A number of witnesses expressed the opinion that veterinary inspectors



Tubercular  
Masses  
(Grapes).

FIG. 32.—Tuberculosis bovis.  
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alone should be employed. On this question we are satisfied that some pathological training is the proper basis upon which to build the knowledge required by a meat inspector, and that, wherever practicable, veterinary surgeons, thus educated, should be employed as meat inspectors. In large towns, where a staff of inspectors is maintained, we do not think it necessary that all of these should be veterinary surgeons, but all meat inspectors should pass an examination and receive a qualifying certificate from a central authority before appointment.

As to the amount and distribution of tubercular disease which justifies the seizure and condemnation of a carcass as unfit for human food, the widest discrepancy prevails in opinion and practice. Chaos is the only word to express the absence of system in the inspection and seizure of tuberculous meat, and it has, in our opinion, become necessary that regulations should be formulated for the guidance of those who are concerned in dealing with this subject.

Entertaining, as we do, the strongest opinion in favour of public over private slaughter-houses, we cannot but recognise in the present arbitrary system of seizure the surest discouragement to the use of public ones. Butchers will seek relief from inspection which they consider unduly strict by using private slaughter-houses, where inspection is either more lenient, or, as in most cases, wanting altogether.

Very strong representations were laid before us on the part of butchers and meat traders, and also on behalf of the agriculturists who supply the butchers, as to the effect of this want of uniformity upon their business. It is obvious, we think, that these complaints are well founded. Producers and traders are making no unreasonable demand when they ask that a recognised standard should be observed, and that meat which, after effective inspection, is pronounced fit for sale in one market, should not be liable to seizure in another because the inspecting authority happens to differ in opinion as to the extent of tuberculosis which may be dangerous.

Of the dead meat imported in vast quantities, the greater part is not inspected till it reaches a public meat market, without the offal and internal organs; and that which is consigned to private establishments generally escapes inspection altogether.

In order to remedy this inequality, and the risk arising from it, we are of opinion that arrangements should be insisted on whereby each animal slaughtered at the port of landing shall be inspected, *together with its own offal*; and further, that "stripping" the pleura of a carcass should be taken as evidence of unsoundness, and be followed by seizure.

We feel that we should be exceeding the limits of our reference were we to dwell on the manifest disadvantage which the home producer suffers in having to submit his carcasses to a more stringent and extensive inspection than the foreigner is exposed to. But this much is clear from the evidence: first, that tuberculosis exists among imported cattle and carcasses; second, that if the rigidity of inspection to which British meat is subjected in some places be justified by the existence of danger therefrom to the consumer, similar danger from imported meat is not provided against under the present unequal system.

Matter of  
Tubercular  
Growth  
(Grapes)

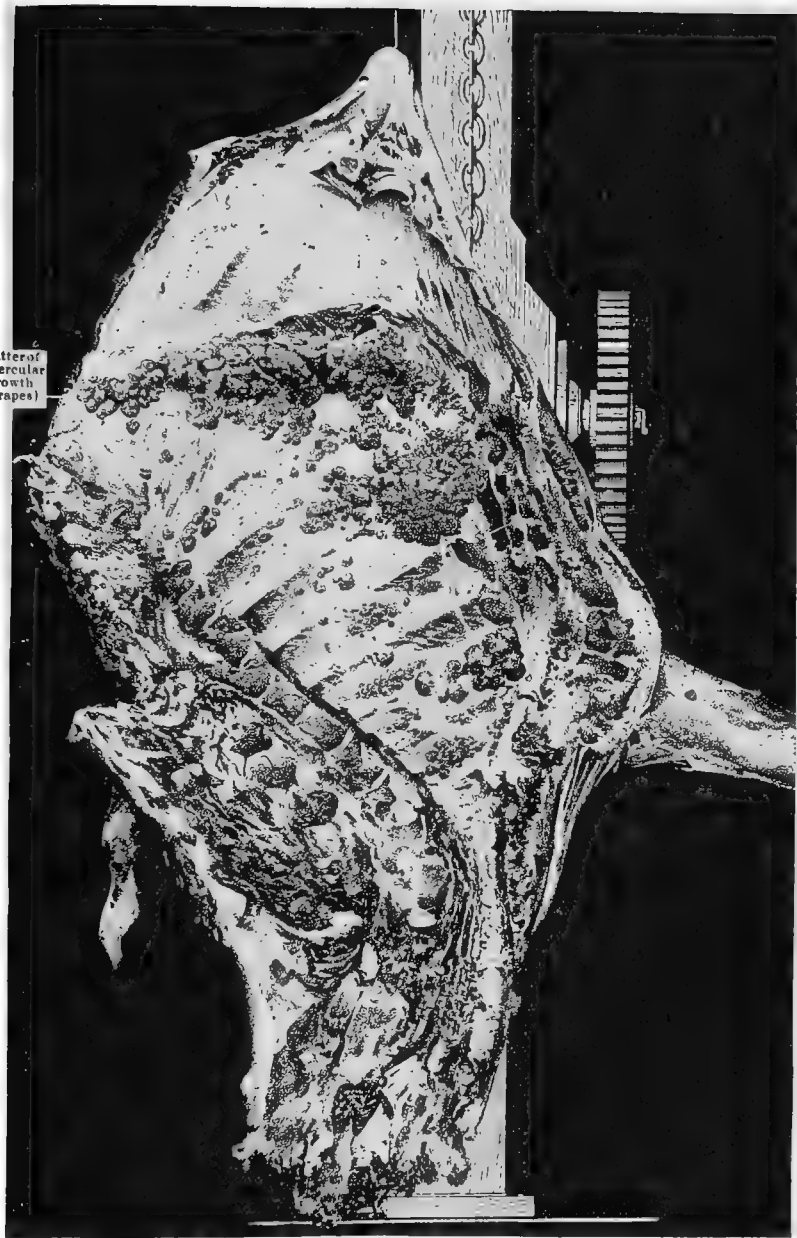


FIG. 33.—Tuberculosis bovis.

We have been favourably impressed with the value of the peculiar institution known in Germany as the Freibank, and alluded to in the last paragraph. It is a department of the slaughter-house where meat of carcasses affected by disease, but not to such an extent as to render it unfit for food, is exposed for sale. It is sold at about half the market rate, in portions not exceeding 10 pounds to each customer, either having been sterilized by exposure to steam for half an hour at a temperature of 100° C., or, where the quality of the meat is considered to warrant it, in a raw state. No butchers, meat salesmen, or restaurant keepers are allowed to purchase at the Freibank; but many poor people, who would otherwise have to go without meat altogether, are able to buy cheap and not unwholesome meat at a very low rate, and the demand is usually found to exceed the supply. Under an efficient system of inspection, we regard the Freibank as the most desirable adjunct to a public slaughter-house, and one that would protect the poor from the unwholesome supplies at present obtained in some of our large towns from the lowest class of butchers.

We are, therefore, of opinion that when, under the certificate of a veterinary surgeon, the sale of milk from a given cow is prohibited, the local authority should slaughter the same, and if, on *post mortem* examination, it appears that the cow was not so affected, the local authority should pay compensation to the extent of the full value of the cow immediately before slaughter. If, on the other hand, the animal be found to have been so suffering, the carcass should be sold by the authority, and the owner thereof should receive the proceeds of the sale.

#### QUALIFICATIONS OF MEAT INSPECTORS.

We recommend that in future no person be permitted to act as a meat inspector until he has passed a qualifying examination before such authority as may be prescribed by the Local Government Board (or Board of Agriculture), on the following subjects:

- (a.) The law of meat inspection, and such by-laws, regulations, &c., as may be in force at the time he presents himself for examination.
- (b.) The names and situations of the organs of the body.
- (c.) Signs of health and disease in animals destined for food, both when alive and after slaughter.
- (d.) The appearance and character of fresh meat, organs, fat, and blood, and the conditions rendering them, or preparations from them, fit or unfit for human food.

#### TUBERCULOSIS IN ANIMALS INTENDED FOR FOOD.

We recommend that the Local Government Board be empowered to issue instructions from time to time for the guidance of meat inspectors, prescribing the degree of tubercular disease which, in the opinion of the Board, should cause a carcass, or part thereof, to be seized.

Pending the issue of such instructions, we are of opinion that the



following principles should be observed in the inspection of tuberculous carcasses of cattle :—

- |   |   |  |   |
|---|---|--|---|
| (a.) When there is miliary tuberculosis of both lungs   | - |  |   |
| (b.) When tuberculous lesions are present on the pleura and peritoneum  | - |  |   |
| (c.) When tuberculous lesions are present in the muscular system, or in the lymphatic glands embedded in or between the muscles | - | The entire carcass and all the organs may be seized. |   |
| (d.) When tuberculous lesions exist in any part of an emaciated carcass   | - |  |   |
| (a.) When the lesions are confined to the lungs and the thoracic lymphatic glands   | - |  | The carcass, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions shall be seized. |
| (b.) When the lesions are confined to the liver   | - |  |   |
| (c.) When the lesions are confined to the pharyngeal lymphatic glands   | - |  |   |
| (d.) When the lesions are confined to any combination of the foregoing, but are collectively small in extent                    | - |  |   |

In view of the greater tendency to generalisation of tuberculosis in the pig, we consider that the presence of tubercular deposit in any degree should involve seizure of the whole carcass and of the organs.

In respect of foreign dead meat, seizure shall ensue in every case where the pleura has been "stripped."

## MILK.

### DISEASES IN THE UDDERS OF COWS.

We recommend that notification of every disease in the udder shall be made compulsory, under penalty, on the owners of all cows, whether in private dairies or those of which the milk is offered for sale.

We recommend that for the purpose of excluding from their districts the milk of cows affected with tuberculosis of the udder, or exhibiting clinical symptoms of the disease, local authorities should be given powers somewhat similar to those of sections 24 to 27 of the Glasgow Police (Amendment) Act, with power to slaughter such cows, subject to compensation under the conditions named in the Report.

We also recommend that powers shall be given to local authorities to take samples and make analyses from time to time of the milk produced or sold in their districts, and that milk vendors shall be required to supply sufficient information as to the sources from which their milk is derived.

At ports where milk and milk products are received from foreign countries, any costs that may be thus incurred in their examination shall be borne by the importers.

## ELIMINATION OF BOVINE TUBERCULOSIS.

We recommend that funds be placed at the disposal of the Board of Agriculture in England and Scotland, and of the Veterinary Department of the Privy Council in Ireland, for the preparation of commercial tuberculin, and that stock-owners be encouraged to test their animals by the offer of a gratuitous supply of tuberculin and the gratuitous services of a veterinary surgeon on certain conditions.

These conditions shall be—

- (a.) That the test be applied by a veterinary surgeon.
- (b.) That tuberculin be supplied only to such owners as will undertake to isolate reacting animals from healthy ones.
- (c.) That the stock to be tested shall be kept under satisfactory sanitary conditions, and more especially that sufficient air space, ventilation, and light be provided in the buildings occupied by the animals.

## TUBERCULOSIS OF THE HORSE.

The horse is seldom affected with tuberculosis, and it is only seen in isolated cases, never attacking many animals in the same stable. An inquiry into the life-history of tuberculous horses has enabled the author to discover that in several instances, where the history could be traced (the animals being still in the hands of the breeders), they had, when foals, been fed on cow's milk either after they had been taken from the mothers, or where the mare had been a bad mother or had died. In these cases the spleen and mesenteric glands were the seats of the lesions, which to a lesser extent involved the liver and bronchial glands.

As in cattle, the symptoms in the horse are generally very obscure, except in one particular, namely, that it is always associated with a more or less rapid emaciation. If the pulmonary organs are involved, there may be a cough, hurried respirations, particularly if excited by exercise or any other cause, and abnormal sounds may be heard in the chest on auscultation. If the contents of the abdomen are the seat of the disease, there may be frequent attacks of colic, with constipation or diarrhoea. Nocard has observed a very pronounced *diabetes insipidus* and irregular variations of temperature, but polyuria is not at all a constant symptom in my experience.



PLATE XVII.



TUBERCULOSIS IN A HORSE FIVE YEARS OLD (MR. HODGMAN'S CASE).

*To face p. 865.*

There is a third form in which this disease affects the horse, and that is what might be called the *vertebral form*.

The premonitory symptoms are gradual emaciation, more or less polyuria, general unthriftiness, and a great desire to lie down continuously, and seemingly to doze.

After a time the animal stands in a stiff position, almost as if he had tetanus; and if an attempt be made to turn him, he either turns *all of a piece* or may fall down. If one tries to put him back, he does it with great difficulty, or may fall in the effort. Later on the stiffness is most marked in the cervical region, and the prominences of these vertebræ can be easily seen standing out from the wasted muscles (*vide* photo-lithograph).

In the great majority of cases in the horse they are isolated ones; but Mr. W. R. Davis, M.R.C.V.S., of Enfield, has placed on record an outbreak of four cases in three years in the same stud.

The tuberculin test may be applied to the horse as well as to cattle, and the appended table will give the reader a good idea as to the efficacy of the test when applied to a suspected tubercular horse:

Temperature prior to and at time of injection	101·5
„ nine hours after injection	- 105·4
„ twelve hours after injection	- 105·2
„ fifteen hours after injection	- 104·4
„ twenty-two hours after injection	103·5

In this case there was a considerable amount of swelling round the seat of inoculation, which remained for eighteen hours (Mr. Hodgman's case).

In the majority of cases observed by the author the spleen and mesenteric glands were the seat of the lesions; the liver, lungs, and bronchial glands being less frequently involved. In some instances the spleen has been enormously enlarged, and studded throughout its substance with grey translucent, yellow-cheesy tumours; whilst in the liver, lungs, and kidneys similar growths are seen, but are caseous in their centres if truly tubercular. Those tumours found in these organs and consisting of white, round, firm succulent tissue, invading the glomeruli of Malpighi and free from bacilli, are not to be considered tubercular; they are those of lympho-sarcoma, having

some degree of organisation ; whereas tubercle is incapable of a higher organisation than the grey nodule, is prone to rapid decay, retrogressive changes commencing in the centre of the nodule, and, the caseous material being the product of the death of the cell elements, the surrounding growth is simply the hyperplasia of the normal tissue, due to the invasion of the bacilli ; in fact, an effort to erect a barrier to the further inroad of the attacking bacilli.

#### TUBERCULOSIS OF THE PIG

is much less frequently met with than in horned cattle, the average, according to statistics, varying from 1·34 to 1·64 per cent. in Saxony, a little more than 10 per cent. in Amsterdam, ·04 at Rouen, ·02 in the Duchy of Baden. In some districts it is never seen, and in Scotland it is so rare that at one time I was inclined to think the pig was immune. I have, however, observed it, and reported the results of the tuberculin test to the Royal College of Veterinary Surgeons ; and in which, one of the most prominent symptoms—not mentioned by authors—was an eruption of small tubercular nodules in the dermoid structures, invading the whole surface of the body.

*Causes.*—The ingestion of tubercular growths, or of tubercular milk. A tuberculous mother has, within my own knowledge, transmitted the disease to her offspring, and in some red Tamworth pigs of my own this was remarkably exemplified. A pregnant sow was sent to me from a celebrated breeder. In course of time she produced a litter, of which the majority did well, but shortly after the young pigs were weaned the sow became rapidly emaciated and was slaughtered ; on *post mortem* she was found to have tuberculosis in an advanced stage, involving most of the organs.

One boar and one sow of this litter were kept, and after the sow became pregnant the boar was sold and not heard of again ; the sow, a very large fine one, gave birth to eight or nine pigs, and shortly afterwards commenced to lose condition very rapidly. An erythematous eruption appeared on the skin, and under the red spots hard nodules soon began to form ; the young pigs thrived for a short time, when some began to fall off rapidly and died tuberculous ; the remaining apparently healthy ones were tested with tuberculin, and all

reacted, and some time afterwards verified the value of the test, presenting signs of the eruption upon and nodules within the skin; rapid emaciation followed, and generalised tuberculosis was found in all. No cough was observed in these cases; the appetite remained fairly good for a time; it then became capricious, and signs of indigestion became apparent, with constipation or diarrhoea. Pallor of mucous membranes, of the tongue, oral cavity, and conjunctiva, as well as of the nose, was a prominent symptom in all the cases; the skin, in addition to the eruption, became very dirty, and the animals soon became miserable objects, with sunken eyes and pendent bellies.

The tuberculin test is the most reliable means of diagnosis, for in the absence of obvious symptoms of tuberculosis its detection becomes otherwise a matter of some difficulty.

The characteristic lesion in the pig is the enlarged sub-maxillary gland, which may be felt between the branching of the lower jaw, and is invariably affected, so that diagnosis in a slaughter-house is rendered easy. On account of the extreme tendency to generalisation of this disease in pigs, it is insisted that the whole carcase should be deemed unfit for human consumption.

*Tuberculosis* is seldom seen in the sheep or goat, but under certain conditions these animals are found not to be immune. Experimentally the disease has been transmitted, particularly by intravenous injection, which sets up an acute, generalised, miliary tuberculosis of a rapidly fatal type, and it is thought that prolonged cohabitation with tuberculous cattle gives the disease to both the sheep and the goat, although they, as a rule, resist subcutaneous and intra-ocular inoculation. The *post mortem* examination of these animals reveals invasion of the lungs, bronchial, mesenteric, and other glands, spleen, &c., and when there is a discharge from the nose the bacilli can be found in abundance.

Care must be taken that actinomycosis, which is not a rare disease in the goat, be not mistaken for tuberculosis.

#### TUBERCULOSIS OF THE DOG.

The dog is much more frequently affected with tuberculosis than is generally believed; one authority found over 4 per

cent. out of 7,000 dogs on *post mortem* examination. It may assume either the acute or the chronic form. A case recently occurred in my practice. The dog had been ill for three or four weeks, and was brought to the College. It died three days after admission. On *post mortem* examination nearly every organ was found to be affected with tubercular neoplasms. They appeared in great numbers on the surface of the pleura, peritoneum, and diaphragm. In the peritoneal covering of the bladder, in the substance of the kidney, and in the liver they showed as minute pearly points, while in the lung their confluence had formed nodes and excavations of various sizes. The heart was studded with nodules, a large one being placed on one of the cusps of the mitral valve. On the base of the heart, and bordered by the pulmonary artery, was an elongated tubercular abscess containing a couple of tablespoonfuls of pus. The spleen was apparently unaffected. The brain and testes were unfortunately not examined. In other cases, however, we have a form much resembling human phthisis, which is characterised by chronic pneumonia and pleurisy, or a chronic asthma and bronchitis. In all cases there is great emaciation and in many there is ascites.

Cats are more frequently affected than dogs; indeed, it is little suspected what a common cause of death consumption is in these animals. The disease in them also often appears as a generalised tuberculosis; sometimes, however, the abdominal viscera and membranes are affected to the exclusion of other parts. Then chains of tumefied glands can be seen along the course of the large intestines, and one or several large tubercular glands in the mesentery. They are extremely hard—cartilaginous, in fact. In the liver, kidney, and spleen the tubercle follicles are as a rule quite small, and appear in sections as a round-celled infiltration, containing, especially in the liver, where they affect the portal space for the most part, beautiful examples of giant cells with their brightly shining zone of nuclei. When in this animal the lung is affected, the formation of vomicae is quite common.<sup>1</sup>

<sup>1</sup> I am strongly of opinion that the presence of dogs, cats and birds in rooms used as living-rooms by people is fraught with much danger, and should not be permitted.—W. O. W.



*Tuberculosis of Birds (Avian Tuberculosis).*—It is maintained by some and contradicted by other writers that the tuberculosis of birds differ in some essentials from that of mammals. Thus Rivolta, Strauss, Mafucci, and other experimentalists have failed to transmit the disease from mammals to birds, whilst Cadiot, Albert, Nocard, and others state that the inoculation is occasionally successful, and that the disease can be transmitted from one to another; and Nocard says: "It is true that the lesions observed in the guinea-pig as the result of the inoculation of avian tuberculosis are widely different, namely, large, red, and soft spleen, absence generally of tubercular nodules in the lungs and liver, but the latter as well as the spleen are crowded with tubercular follicles rich in bacilli, and a few transmissions from guinea-pig to guinea-pig are all that is necessary to enable these lesions to reproduce the type so well described by Villemin."

Lastly, the same writer says: "It must be remembered that the tubercle bacillus so resistant to all causes of destruction may, however, experience profound modifications by means of successive passages through the organs of divers species of animals. But if the modifications which it undergoes as the result of numerous transmissions through birds are profound enough to make the bacillus of avian tuberculosis a peculiar variety of the bacillus of Koch, they are not enough, in my opinion, to make these bacilli two distinct species."

The avian bacillus is longer than that of mammals, grows more rapidly and more readily on cultivation media, appearing as thick, moist, luxuriant spots, which preserve their vitality longer than those from man. They grow at a higher temperature—109° F.—whilst those of the mammal cease to grow at 104° F.

To prevent the spread of avian tuberculosis it will be necessary to kill all the birds in the infected yard; the diseased ones being cremated or buried deeply in the ground, their bodies surrounded by a layer of some antiseptic, the healthy birds of course being disposed of for food as quickly as possible. All the wood-work of the hen-houses should be destroyed, the walls scraped and whitewashed, the wash to contain carbolic acid, the excrement removed and burnt with the wood-work, and on no account should birds from an infected run be sold except for slaughter.

## TREATMENT OF BOVINE TUBERCULOSIS.

If the disease has passed beyond the very earliest stage, it is a waste of time and money to treat animals suffering from tubercular consumption. It is far better to slaughter and make the best of them; and in all cases it is better to make the animals fit for the butcher by ceasing to milk them, giving fattening food such as oil-cake and good hay, avoiding grasses and roots, as they tend to produce indigestion, diarrhœa, and an acid condition of the digestive apparatus, and by administering cod-liver oil in such quantities as the animal may digest and assimilate—say, from six ounces to half a pint daily; if purgation is not induced, the latter quantity. The oil is best given mixed with lime-water, and small doses of oil of turpentine may also be added with advantage, particularly if there be a tendency to indigestion, tympanites, or diarrhœa.

The following curative or preventive agents have been recommended: iodine (Williams), mercuric chloride (Herron), eucalyptus oil (M. Ball), salicylic acid (Griffiths), creasote (various), inhalation of turpentine, hydrofluoric acid, &c. (various); but so far these have proved uncertain. Paraffin has also been recommended, but the results do not warrant the continuance of any medicinal treatment.

In 1901 Von Behring published his method of immunising cattle against the disease. Some years previously he had inoculated a guinea-pig with sputum from a human subject, and had continued the cultivation.

These bacilli so obtained were dried *in vacuo*, and to them was added a few drops of glycerine, and the two were well rubbed together in a special mortar. This mixture was then gradually emulsified by the slow addition of normal salt solution, and 1.5 parts of sodium carbonate in each 1,000 parts; thus every 2 c.c. contained 4 milligrams of the dried bacilli.

To afford immunity, two intravenous injections are necessary, the first to contain 4 milligrams of dried bacilli, and at the expiry of twelve weeks the second injection, consisting of 20 milligrams. The immunity lasts for about a year. Quite recently Von Behring has produced a new preparation named "tuberculase," which is said to be more certain in its action.

## STRUMOUS ABSCESSSES.

In the *Provisional Nomenclature of Diseases* adopted by the Royal College of Physicians, London, tubercular diseases are considered under two heads, namely—(1.) Scrofula with tubercle, sometimes a concomitant of internal tuberculosis; and (2.) Scrofula without tubercle. This latter condition is witnessed in the sheep, in which the true tubercular nodule is rarely developed. This ruminant, however, is prone to suffer from strumous scrofulous abscesses in the submaxillary, facial, and parotidean regions, sometimes commencing in the connective tissue about the jaws, neck, and face, and involving the lymphatic glands (strumous adenitis), and known as “cruels” by Scots farmers.

These abscesses often appear, without any premonitory signs of ill health, as hard swellings, which sooner or later suppurate imperfectly, or burrow deeply in all directions; at first they are not painful, but often become very much so, and may multiply rapidly, appearing between the jaws, on the neck, about the eyes, lips, and nostrils, and occasionally upon other parts of the body, causing swelling, preventing the sheep from feeding, and rendering the breathing difficult, and snoring. In some instances swelling of the face and discharge from the nose are observed previous to the development of the tumours.

Young animals, particularly young fast-growing tups, if exposed to cold, are especially subject to this disease, but older animals of both sexes, as well as lambs, are not exempt.

If these abscesses are opened, the quantity of pus contained within them, not always commensurate with their size, is generally thick, and flows tardily, but afterwards becomes thin, and more or less ichorous; it is surrounded by a thick sac of low fibrous material, like the walls of an old ulcer, and sinuses are often found running from the cysts into the surrounding structures. Microscopically examined, the pus is found to consist of ordinary pus cells, rather shrivelled in appearance, but showing little or no tendency to caseation, and the tubercular bacilli are absent, but the pus may be loaded with the cocci of suppuration. In many instances the tumours undergo little or no change for two or three years, the sheep seemingly suffering no inconvenience; but in other cases the

animal loses flesh rapidly, the wool falls off in patches, symptoms of fever manifest themselves, a cough is now and then heard, and the sufferer dies from exhaustion, anæmia, and sometimes dropsy. The flesh in this advanced stage is pale and watery, but if an animal be slaughtered in the earlier stages it is fairly good.

The *post mortem* appearances are often quite local, merely collections of pus in the various thick-walled abscesses observable before death; the pus will, however, be found to have burrowed from the abscesses in various directions into the surrounding tissues. Now and then some degree of ulceration of the nasal mucous membrane may be detected, and abscesses found in the lungs, but these two latter conditions are by no means constant.

Strumous abscesses in sheep, or the conditions which lead to their formation, do not seem to be hereditary, as the stock of many tups affected remain quite free from the disease, provided they are not exposed to that cause—namely, cold—which induces it in the progeny of those which have never been affected.

*Treatment.*—The cause or causes being removed, it will be necessary to open the abscesses as soon as pus can be detected in them, a matter of some difficulty, as their walls are very firm and unyielding; however, if by firm pressure with one hand, and manipulation of the tumour with the other, the pus can be felt fluctuating slightly, the operator need not hesitate, but make a bold incision, press out the semi-fluid matter, and dress the wound with some digestive, such as turpentine liniment. The parts are to be kept clean, dressed antiseptically daily; the wounds prevented from closing too quickly, and the animal fed liberally. A small quantity of sulphate of iron in the food often assists the recovery. A varying degree of swelling may exist for a time after the wounds have healed.

Abscesses about the throat in cattle are often supposed to be tubercular, when in reality they are due to the actinomyces.—(See Actinomycosis.)

#### PREPARATION OF TUBERCULIN.

For diagnostic purposes this preparation of the products of the development of the bacillus in culture media has proved of

incalculable value, experiments having been made by scientists in almost all parts of the world, and with the almost invariable result of inducing a reaction in diseased animals only, healthy ones, or those free from tuberculosis, remaining unaffected.

In France a strong tuberculin is prepared as follows, but it must be diluted prior to use: A pure culture of tubercle bacillus is inoculated into broth containing five per cent. of glycerine, and this is kept at a temperature of about 98° F. in an incubator for six weeks. The broth, now teeming with bacilli and rich in their products, is sterilised by heating to 230° F. in the steam steriliser. This kills the bacilli, while their products remain unaltered. It is now evaporated until one-tenth only of the original volume remains, and is filtered.

In England it is now made by growing the organisms for twenty-eight days, or until a thick uniform scum covers the whole surface of the broth at a temperature of 38° C.—i.e., 103° F. There is some difficulty in getting a culture on the surface, by which means only a good toxin can be obtained; but this is accomplished after some time and several efforts. The growth is then sterilised and filtered. The filtrate is used as tuberculin without any further condensation.

The preparation of mallein is accomplished in practically the same way. It may be noted that in both tubercle and glanders the best medium in which to keep stock cultures is potato.

The action of mallein and tuberculin is not properly understood, but the reason of its affecting animals suffering from the specific diseases of which these products are the toxins is that the excess of such products being inoculated into a system already charged with similar substances causes a local and general reaction which could not take place in a normally healthy animal, whose system would be capable of dispersing it without showing any constitutional trouble. We know that an animal suffering from either chronic glanders or tubercle has periods of illness and comparative freedom from any symptoms. When such an animal's temperature suddenly rises, it is possibly due to the liberation of an excessive amount of toxin from organisms growing freely in some encapsuled lesion. The inflammatory envelope becomes patent, and allows of the flooding out of toxin, and probably also of

the microbes themselves, into a circulation that is already charged with as much of the bacterial products as it can stand without showing a reaction. The inoculation of mallein or tuberculin will create an analogous condition, and so explains the phenomenon. The extracellular products known as mallein are undoubtedly a toxin, but it is very improbable that they are altogether the same as those that are produced in the animal body. In accordance with Wright's theory of opsonins, the explanation is that the fluid of the blood contains a subnormal amount of the specific opsonin required for the proper preparation of the tubercle or glanders organisms, so that they are continually getting ahead of the phagocytic power of the leucocytes to cope with them, and it is at such periods, when the bacteria have become so overabundant, with a consequent increase in toxin formation, that acute symptoms supervene. This flooding of the system, as it were, with bacteria and their products acts as a stimulant to the production of opsonin, and so defeats itself. An artificial stimulation may be accomplished in the same way by the administration of bacteria in the form of vaccine, by the inoculation of which, intra-, and possibly some extra-, cellular toxins are introduced. By the proper administration of such substances, an animal's opsonin content may be so increased as to induce a cure of the disease. The application of a tight bandage (Bevis bandage) to a local lesion of tubercle will induce a stagnation of the lymphoid fluid, which will become bathed in the bacterial products. On removal of the bandage, the excess fluid will be reabsorbed into the circulation, and act as would a vaccine. In this is constituted the danger of surgical interference in the treatment of microbial local lesions. The cutting of surrounding vessels and tissues facilitates the inoculation into the system of bacteria, and their products convey a generalised infection which, but for that interference, would have remained encapsuled in the fibrous or inflammatory zone which the irritation of the organisms has established.

It will be seen from this short explanation how that the separated inoculation of tuberculin will so stimulate the protective powers of the blood that the reaction will be quicker and more easily respond to the stimulus the oftener it is

applied, and that each time this reaction, being more in the nature of a response to a healthy stimulus, will be more evanescent in character, simply because the animal economy is in a more efficient state of meeting it. It must not be concluded from the above that mallein and tuberculin are the same as a vaccine—*i.e.*, actual bacteria. They are only the extracellular products of these bacteria, and are useless to stimulate the production of antibacterial bodies. They can only stimulate those conceptions of Ehrlich which are capable of dealing with toxins as apart from bacteria (*vide* chapter on Ehrlich's theory).

#### METHOD OF INOCULATION AND EFFECT OF TUBERCULIN.

Upon the discovery of tuberculosis in a herd, all animals in contact should be examined, the temperature of the animals to be operated upon should be taken and carefully noted for a day or two before the inoculations are made. The seat of operation should be the lower parts of the neck or behind the elbow. The part is to be well washed with a five per cent. solution of carbolic acid and the syringe disinfected with the same solution.

Taking up a fold of the skin, the needle is pushed into the loose subcutaneous tissue, which in these positions is abundant, and about 40 minims of the tuberculin injected. The temperature is to be taken ten, twelve, fifteen, eighteen, and twenty-one hours after the injection. The following are conclusions arrived at after very extensive trials, and published in the "Annales of the Pasteur Institute":—

1. Tuberculin has a specific action on tuberculous bovine animals, which is expressed by a considerable rise in temperature.
2. The injection of a strong dose gives rise in tuberculous animals to an elevation of temperature of between 2 to 5 degrees Fahrenheit.
3. The same dose injected into non-tuberculous animals does not ordinarily produce any appreciable febrile reaction.
4. The febrile reaction appears most frequently between twelve and fifteen hours after the injection, sometimes at the ninth hour, very rarely after eighteen hours: it

always lasts several hours. It has been found that after one, two, or more injections of tuberculin that the reaction takes place more quickly and is more evanescent, so that all traces may have passed off in a few hours. This has been taken advantage of by unscrupulous dealers, and it is therefore necessary to commence taking the temperature at once, and continue to do so up to the eighteenth hour if necessary.

5. The duration and the intensity of the reaction are not related to the number and the gravity of the lesions; it seems even that the reaction may be more decisive in the case where, the lesion being very limited, the animal has conserved the appearances of health.
6. In the subjects gravely affected by tubercle, and especially in those which are fevered, the reaction may be little marked or absolutely wanting.
7. It is prudent to take the temperature of animals morning and evening for several days before the injection, because it may happen that from trifling and temporary affections — congestion and the like — there may be fluctuations of temperature productive of serious error. For these animals it is necessary to put off the injection.
8. In certain tuberculous animals, not fevered, the reaction consecutive to the injection of tuberculin does not reach more than  $1.4^{\circ}$ ; still, as experience shows that in perfectly healthy animals the temperature may undergo variations of  $1.4^{\circ}$  or more, only reactions above  $2.4^{\circ}$  should be allowed to have any real diagnostic value. All animals in which the injection is followed by a rise of  $1\frac{1}{2}^{\circ}$  to  $2\frac{1}{2}^{\circ}$  should be considered as suspect, and ought to undergo a new injection after the lapse of about a month.

Professor Dr. A. Eber, Leipzig, on tuberculin testing, says:

- (a) For young cattle up to six months of age:

In those whose temperature does not exceed  $104^{\circ}$  F., any rise about  $104^{\circ}$  F. is to be regarded as a reaction, provided the difference before and after injection be not less than  $0.9$  F.



(b) Cattle above six months :

- (1) No animal whose temperature is above  $103.1^{\circ}$  F. to be tested.
- (2) Rises of temperature to  $103.1^{\circ}$  F. to be regarded as non-suspicious.
- (3) Every animal whose temperature before inoculation is below  $103.1^{\circ}$  F., and rises above  $104^{\circ}$  F., be regarded as suspicious.
- (4) Rises between  $103.1^{\circ}$  F. and  $104^{\circ}$  F. to be accounted as reaction, provided the difference before and after amounts to at least  $1.8^{\circ}$  F.
- (5) Similar rises, but not up to  $1.8^{\circ}$  F., to be regarded as doubtful, and to be especially considered. In true reaction there is a special temperature curve—up, pause, and fall.
- (6) In ordinary testing all should be regarded as suspicious whose temperature rises above  $103.1^{\circ}$  F., providing the rise is at least  $0.9^{\circ}$  F.

Crookshanks and Herroun have separated a ptomaine and an albumose from the crude glycerine extract of cultures, which, when ejected hypodermically into tubercular guinea-pigs, induces a rise of temperature, and its effect on tubercular glands in the cases associated with rise of temperature was to render them well defined, indurated, and painful. Other experiments have been made to determine the action of the amide group of organic substances upon the economy in health and in tuberculosis. In a report by Professor Samuel G. Dixon, M.D., and W. L. Zuill, M.D.D.V.S., Academy of Natural Science, Philadelphia, it is stated that, in endeavouring to discover the true nature of the active principle of tuberculin, a crystalline substance was produced that at once suggested the amide group,—allantoin, glycozin, tyrosin, kreatin and kreatinin, taurin, cystin, &c. They determined to make use of kreatin, which was at hand, and injected a small quantity into tuberculous and healthy small animals, and afterwards by Zuill into cattle, with results resembling those obtained by tuberculin. Zuill states that the action of kreatin upon tubercular tissue is intensely energetic, causing its rapid necrosis, giving it the appearance of having undergone a cystic de-

generation. Experiments with taurin also caused elevation of temperature.

Mr. George N. Kinnell, M.R.C.V.S., Pittsfield, Mass., reports (1895) that more definite results are obtained by using smaller doses of tuberculin. He says: "By a smaller dose I mean two-thirds of a minim; a large dose, the ordinary one from three to five minims—of Libbertz's Tuberculinum Kochii. The small doses will not cause reaction in the earlier stages, or where the disease is but slightly advanced, but will do so in advanced cases where a large dose has failed to cause elevation of temperature."

We strongly desire to impress upon the reader that neither tuberculin nor mallein is of any particular value, and cannot be relied upon when injected into animals suffering either with or recovering from other diseases, more especially those of the Pasteurella order.

At the International Congress on Tuberculosis, held in Philadelphia this year (1908), Dr. Melvin (Chief of the United States Bureau of Animal Industry) considered the economic aspects of tuberculosis in food-producing animals. Statistics of the United States Federal Meat Inspection for the year ending June 30, 1908, concerned 53,973,337 animals, or more than one-half of all those slaughtered for food in the country. They showed the following percentages of tuberculosis: Adult cattle, 0.961; calves, 0.026; hogs, 2.049; sheep and goats, 0. The proportion of tuberculosis was probably higher in animals slaughtered without inspection. Reports of tuberculin tests made in the fifteen years from 1893 to 1908 with tuberculin, prepared by the Bureau of Animal Industry, show that of 400,000 cattle tested—mostly dairy cattle—there were 37,000 reactions, or 9.25 per cent. From these two classes of statistics the author concluded that on an average about 10 per cent. of the milch cows, 1 per cent. of other cattle, and 2 per cent. of the hogs in the United States were affected with tuberculosis, the average percentage for all cattle being estimated at 3.5. The accuracy of the tuberculin test had been confirmed in a remarkable manner by *post mortem* examination. Out of 23,869 reacting cattle slaughtered, lesions of tuberculosis were found in 23,585, a percentage of 98.81. The loss on animals in which tuberculosis was found

in the Federal Meat Inspection was estimated at \$2,382,433 annually, and if the same data were applied to animals slaughtered without Federal Inspection the loss would amount to \$4,354,855. The stock of animals was also depreciated in value because of tuberculosis. Assuming that living tuberculous milch cows were annually depreciated to the extent of one-tenth of what the loss would be if they were slaughtered, other cattle one-third and hogs one-half, the total annual depreciation amounted to \$8,046,219. The annual loss in decrease in milk-production was estimated at \$1,150,000, and there was also some loss from impairment of breeding qualities. Taking all these items into account, the aggregate annual loss from tuberculosis among farm animals in the United States was estimated at not less than \$14,000,000.

Dr. E. C. Schroeder, of the Department of Agriculture, Washington, read a paper on his investigations, showing that about 40 per cent. of cattle, apparently healthy, and known to be tuberculous only because they had reacted with tuberculin, intermittently passed tubercle bacilli with their fæces. When a number of such cattle were kept under continued observation, the percentage that expelled tubercle bacilli per rectum was found to be double in about eighteen months; and both the frequency with which bacilli occurred and the number found in individual animals also showed an increase. Among twelve cows, bacilli in the fæces were at first found in five, but before the end of two years the number had risen to ten. The proof that the acid-fast bacilli demonstrated by microscopic examination actually were tubercle bacilli was established by (1) producing fatal generalised tuberculosis in guinea-pigs inoculated with the fæces; (2) causing fatal generalised tuberculosis in hogs by feeding them with the fæces; and (3) causing fatal generalised tuberculosis in guinea-pigs by inoculating them with milk soiled with small quantities of such fæces, and with butter made from such soiled milk. Pure cultures of tubercle bacilli, isolated from the infected guinea-pigs, caused fatal tuberculosis in cattle on subcutaneous inoculation. Dr. Schroeder further observed that with rare exceptions commercial milk could be shown on examination to be contaminated with cows' fæces, and he concluded that, considering the wide prevalence of tuber-

culosis among cattle, the presence of fæcal material in milk was frequently associated with the presence of virulent tubercle bacilli. Cream and butter were also exposed to this danger.

Dr. Hermann M. Biggs, General Medical Officer, New York, described what had been done in New York City with a view to the control of tuberculosis. Following an extensive campaign of education, the Board of Health in 1894 classed pulmonary tuberculosis as an infectious disease, and required all hospitals and public institutions to report all cases under their charge. Private physicians were asked to do the same, and the Board of Health established a diagnosis laboratory, where sputum was examined for them free of charge, the only stipulation being that the physician should furnish the name and address of the patient. In the course of time it became recognised by the public that the Board of Health was interfering neither with the work of the private physicians nor with the right of the individual, so that in 1897 the Board was able to adopt a regulation requiring even private physicians to report their tuberculosis cases to the health authorities. At the present time the basis of the tuberculosis campaign in New York City consisted in the compulsory notification and registration of all cases of the disease. The reported cases fall into two large groups: (1) Those reported by private physicians as being under their care, and (2) those reported as not being under such supervision. If the consumptive was regularly under the care of a private physician, no further action was taken by the Board. If, however, he were not, or if he were receiving charitable medical advice, then all objection to the visitation and the supervision of the case by the Health Department was removed, and, in fact, the Department was required to intervene. In this latter class of case the health authorities did their most effective work. The consumptives were visited in their homes by trained nurses or by physicians. If country air would benefit the patient, the Department of Health offered to take him to its country sanatorium at Otisville or to refer him to the State sanatorium at Raybrook. If the patient was able to be about, and could not leave the city, he was offered free medical care at a city tuberculosis dispensary; if he were bedridden, and in a decent home, he might receive medical care at the hands of the

Board's physicians, assisted by trained nurses ; if he needed hospital care, he was sent to a special hospital ; and, finally, if the consumptive was filthy in his habits, and so situated that he was a menace to those about him, the Board could forcibly remove him to its special tuberculosis hospital on North Brother Island. Owing to the multifarious nationalities populating New York, the anti-tuberculosis campaign had been far from easy. Millions of circulars in a score of languages had been supplied and distributed by the Health Department, and had instructed the people as to the nature of consumption and how to avoid contracting it. Lectures had been given in the public parks and schools. Exhibitions, showing the right and wrong way to live, had been held in various parts of the city, and had been found to be even more effective than words. "Don't spit" notices had been placed in all the public conveyances, railway-stations, &c., and flagrant offenders had been arrested and fined. The result of the campaign had been that the death-rate from tuberculosis had dropped from 4.42 per 1,000 in 1886 to 2.41 per 1,000 in 1907.

Professor Calmette stated that after numerous experiments he had prepared for therapeutic use a particularly active and relatively pure tuberculin called Tuberculin CL, which could be introduced intravenously into the body of a healthy animal in large doses, without producing any elevation of temperature. In this respect it possessed an advantage over all other tuberculins. As much as 50 centigrams could be injected at a single dose into the jugular vein of a healthy bovine without producing any reaction ; if, however, the same injection, in the same dosage, were repeated three times, at intervals of six to ten days, the animal, in from five to twelve hours after the third injection, reacted by a rise of  $1.8^{\circ}$  C. to  $2.50^{\circ}$  C., just as though it were tuberculous. A further consequence of such repeated inoculations was that the animal acquired a high degree of resistance to artificial tuberculous infections. The intravenous injection of a dose of virulent bovine bacilli, sufficient to produce without fail an acute and fatal miliary tuberculosis in control animals in from four to six weeks, merely produced in an animal prepared by previous injections of tuberculin a chronic and slowly progressing tuberculosis.

The author's tuberculin was prepared by centrifuging *in vacuo*, at a low temperature, entire cultures of the bovine bacillus. The product was then filtered, in order to separate the bodies of the bacilli, precipitated three times with alcohol and ether, redissolved in water, and dialysed until all the precipitants and salts had been completely eliminated. The colloid substances which remained on the dialyser were then precipitated once more with alcohol and ether, and dried *in vacuo*. When the activity of this tuberculin was tested by direct inoculation into the brain of a healthy guinea-pig, it was found that 0·0008 milligram sufficed to kill the animal, whereas almost ten times as much of Koch's old tuberculin was required to produce the same result. The greater activity of the new preparation was attributable to the avoidance of heat during its manufacture and to the absence of all chemical treatment except precipitation with alcohol and ether. Professor Calmette stated that his Tuberculin CL was very well borne by tuberculous patients, and, whilst not curing tuberculosis any more than any other tuberculin, it evidently delayed the progress of the disease, and endowed the organism with resistance to the infection. It was recommended to begin with a very small dose—one-thousandth of a milligram—and to increase it gradually by fractions to one to three thousandths, hundredths, and tenths of a milligram, administering the injections at intervals of ten to twelve days. At the same time, an effort should be made to obtain a constant increase of the opsonic index over the index of the previous inoculation, or at least to maintain it at the same level.

#### SUGGESTIONS FOR SUPPRESSION OF TUBERCULOSIS.

1st. *For dealing with Non-Breeding Cattle.*—When tuberculosis is suspected in an animal, it and all in-contact cattle should be subjected to the tuberculin test, and that all in which the reaction followed, if in good condition, be slaughtered, and the beef, if found fit after careful examination by a duly qualified veterinary inspector, be sold for human consumption. That the animal be valued prior to slaughter, and that the difference between the value of a milch cow as beef be made up by the Government as in pleuro-pneumonia, or perhaps divided between Government and owner.

slaughtered and their carcasses at once destroyed, no compensation being given, and that neglect to report such cases to the proper authority be considered an indictable offence.

That all animals in which the tuberculin reaction indicated that they were affected, but apparently showing no signs of ill-health, if not fit for slaughter, should be prepared for the butcher and slaughtered when fit, being in the meantime removed and isolated from those in which no reaction had taken place. That the healthy stock be again tested and the same rules applied.

*2d. For dealing with Breeding Stock.*—In dealing with breeding stock, I can do no better than quote from Nocard, who says: "All those engaged in agriculture, in breeding, rearing, feeding, or fattening, ought to carry out each for himself the prophylaxis of the disease. Each of them is directly interested in it. The methodical use of tuberculin, by denouncing the sick animals at the outset of the disease, permits one to isolate them, and to protect the sound animals from all danger of contamination.

As the young animals mostly escape the infection, breeding would not be seriously interfered with, and the vacant spaces would be filled in a few years. Of course, a farm once made healthy ought to be protected from re-infection. To effect this it would be sufficient to introduce into it no new animals without having them previously tested with tuberculin.

Professor Bang of Copenhagen has a similar scheme, and has, according to his last report, carried it to a successful issue. He proceeds as follows:—

The whole herd is tested with tuberculin, and thus divided into a healthy and a tuberculous section, which are separated from one another, and have separate attendants. The healthy section is tested every six months with tuberculin, and any animals which react are at once removed to the tuberculous section. Those animals of the tuberculous section which are obviously affected are got rid of; but those which are apparently healthy are kept and used for breeding purposes as long as may be convenient, and as they will generally be fattened for slaughter before the disease is far advanced, the total condemnation of their carcasses as butchers' meat will not, as a rule, be necessary. The calves of the tubercular section are removed to

the healthy section immediately after birth, and are fed for the first day on colostrum, which has been heated to 65° C.—149° Fahr.—and subsequently on boiled milk, this boiled milk being from tuberculous cows.

At first these calves were kept in separate boxes, and only added to the sound section when they had successfully undergone the tuberculin test, but he now thinks that they may with safety be removed to the sound section immediately after birth, and wait till the time of the general half-yearly testing with tuberculin.

In carrying out the above, I think the Government might safely devote an annual sum of money to assist cattle-owners in suppressing so great and increasing an evil as tuberculosis; and, being of opinion that the schemes are tangible and reasonable, I would now suggest that tuberculosis be included in the statutory schedule of infectious diseases, and that compensation be paid to the owners of cattle affected with tuberculosis when such cattle are slaughtered in the public interest.

The eradication of tuberculosis is a question of absorbing interest, but difficult of practical realisation. There should certainly be more endeavour made to limit the progress of the disease amongst cattle, and undoubtedly the first attempt should be made with milch cows. The amount of disease that it is possible to disseminate in milk is incalculable. The present indiscriminate regulations, which are applicable in some places and not in others, are useless from a general practical view. It would appear that nothing can be accomplished until the State will compensate farmers for the loss that would be occasioned by compulsory slaughter, as is done in the United States, and this method should be adopted. The initial cost would be heavy, but it would not be a heavy recurrent one, and the benefits would be enormous. By the use of tuberculin, diagnosis may be made quickly, and in the early stages, when by slaughter the greater part of the meat would be good. This, in combination with the increasing number of phthisical homes and retreats for consumptives, would in a few years cause a great diminution in the ravages of this scourge. Even with the paltry measures now adopted in some towns in dairy inspection, great improvements are acknowledged, but it is unfair to ask the farmer to do even as much as he is doing without compensation.



## CHAPTER XXXVI.

### CONTAGIOUS DISEASES—*continued.*

#### TETANUS (LOCKJAW).

TETANUS is usually described as a powerful and painful spasm of the voluntary muscles, which is long-continued and uncontrollable. The spasm of the muscles is that of rigid contraction, and from its constancy and non-intermitting character it has been termed tonic. It is due to the presence of the bacilli of tetanus in a wound (see fig. 34, p. 388). Of all the domestic animals, the horse is most liable to tetanus. It is but rarely seen in the ox or dog.

Tetanus may result from a very trivial injury, although it is most likely to do so after a severe laceration or puncture, more especially when nerves are injured. Wounds of the feet and joints, although giving rise to a high degree of irritative fever, seldom favourably harbour the tetanus bacilli, and in my experience wounds in the region of the quarters, thighs, and fore-arm, more especially if the great nerves of those parts are injured, are those most liable to encourage its growth.

The operations which are most commonly succeeded by tetanus are docking, castration, the insertion of setons, and in one instance which fell under my notice a moderate blister to a fore-leg was a precursor of tetanus.

Tetanus is rarely seen in certain districts. Mr. Cartwright, of Whitchurch, informed me that he had never seen a case of tetanus in his district, although he practised there for forty-five years; and during the ten years I practised in Bradford I saw but two cases. In other districts of the country tetanus is exceedingly prevalent.

Tetanus is occasionally seen as an enzootic disease, simul-

taneously attacking several animals in the same district. During the summer of 1858 I witnessed ten cases in a fortnight. Some writers on veterinary surgery state that tetanus is more apt to prevail in cold than in hot weather. My experience is contrary to this, and that it is mostly during warm weather that the disease prevails to any extent, although isolated cases of it occur at all times of the year.

There are several varieties of symptoms of the disease, and the word tetanus is made use of to denote it generally. As a generic term, it comprehends all the varieties, but when not used in this sense it implies that the disease involves all classes of muscles equally. When the muscles of mastication are alone involved, it is called *trismus*. When it chiefly affects the superior cervical and dorsal muscles, causing the head to be elevated and the spine curved downwards, it is called *opisthotonos*. When the muscles of one side are affected, it is called *tetanus lateralis* or *pleurosthotonos*; and in other cases—rare even in the human being—the inferior muscles are chiefly affected, the chin drawn towards the breast, the spine curved backwards, the disease is named *emprosthotonos*. In the lower animals, trismus, with opisthotonos, is generally met with. I have seen a modified form of tetanus lateralis, but the other form—namely, emprosthotonos—is, I think, unknown in either horse or ox.

Tetanus, whatever be the variety, may be acute, subacute, or even chronic. The acute is that which is most common, and most fatal; it has a tendency to involve the whole frame, and to destroy life by arresting the respiratory movements. Amongst the uncommon variety of causes of wounds which may permit of the entrance of the bacilli of tetanus, in addition to external wounds, I have observed worms in the stomach and intestinal canal, collections of sand in the large intestines, and uterine irritation after abortion.

Tetanus follows injuries, whether inflicted surgically or otherwise, in an indefinite but limited period of time; but usually the occurrence of the spasm is not observed until the wound is nearly or quite healed. Neglect in the treatment of, the presence of a foreign body in, or the application of irritating medicaments to, a wound, is apt to favour tetanus.

*The Symptoms of Tetanus.*—In the earliest stage there will

be a stiffness of the muscles near the seat of the injury ; if a limb is wounded, the animal will move it with difficulty ; the stiffness spreads over the whole body ; the animal will begin to champ his jaws and grind his teeth, the power of opening the mouth gradually diminishing, until the jaws become firmly locked. There is often a flow of saliva from the mouth and a collection of froth upon the lips. The breathing now becomes accelerated, the nostrils dilated, the nose protruded, the membrana nictitantes pushed more or less over the eyes, which are withdrawn within their sockets, best observed when the patient's head is elevated or he becomes excited. If the animal be suddenly disturbed, the superficial muscles will be seen to twitch or tremble ; the eyeballs, convulsively withdrawn within the orbits, causing the patient to show the white of the eye at every convulsive retraction ; the tail is suddenly elevated, and is maintained in that position by an irregular clonic spasmodic action of the levator muscles so long as the excitement continues.

At first the pulse is not much affected, and in all but the most severe attacks it continues undisturbed for two or three days. It has a hard, incompressible character, however, and as the disease advances it becomes accelerated, harder, and more incompressible. When the spasm becomes general, the position of the various parts of the body is regulated by the action of the more powerful muscles. The limbs are extended, flexion of them is performed with difficulty, and the patient stands with outstretched limbs, and there is a hyperæsthetic condition of the skin. The course of the *levator humeri* can be easily traced, and the contraction of this, and other muscles which act upon the superior part of the cervical region, cause the neck to assume the appearance of what is termed "ewe neck." The peristaltic motion of the bowels is stopped ; the urinary bladder firmly contracted ; a dry, husky cough comes on when the animal attempts to swallow, and the act of deglutition is performed with a difficulty which increases from day to day. The muscles of the abdomen are rigid ; the belly looks small and hard ; the intercostals act imperfectly ; and when the diaphragm becomes involved, the breathing is performed with very great difficulty.

Although the spasm of tetanus is of the tonic or persistent

kind, there are exacerbations of a clonic intermittent character; and the whole course of the disease is marked by paroxysms of great severity if the animal be subjected to meddling attendance, strong light, or rustling noises. In a modified light, and when the animal is kept quiet, the spasms are usually diminished and the exacerbations much milder.

For many years it was suspected by many English veterinarians that tetanus was both a contagious and infectious disease. The contagiousness of tetanus has now been conclusively proved, the disease having been transmitted by inoculation, even with tetanin (without bacilli) obtained from the spinal cord of tetanic subjects.

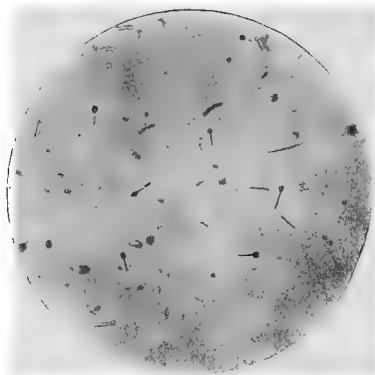


FIG. 34.—Bacilli of tetanus.  $\times 3500$ .

The contagiousness of the disease is due to a micro-organism—the *Bacillus tetani*—described by Nicolayer, 1884, and by Rosenbach, which gives rise to the formation of a toxin termed *Tetanin*. This organism is longer but narrower than that of anthrax, having one enlarged spore-bearing end. Nocard has demonstrated that dried blood and pus scraped from instruments employed in castrating horses, all of which had died from tetanus, caused the disease when inoculated into rabbits. The *Bacillus tetani* is also found in garden mould, and it is possible to explain the non-existence or rarity of the disease in some districts, and its prevalence in others, upon the assumption that the organism common to the soil gains entrance to the animal economy.

Bearing these conclusions in mind, the practitioner will not fail to see the probable and beneficial effects of germicide treatment, both locally and generally. Weak sublimate dressings to the wound, tincture of iodine, iodide of potassium, carbolates, salicylates, and biborates internally.

In *post mortem* examinations of tetanus I have invariably found the nerves leading from the injured parts to present some signs of inflammation; the *neurilemma* more vascular than natural, the vessels of the spinal cord engorged, and the sub-arachnoid space to contain some effusion.

*Treatment of Tetanus.*—Suppose the disease to be caused by docking or neurotomy, the first question to be considered is whether a portion of the stump of the tail or the end of the nerve should be removed or not. The nervous irritation is due to the nervous fibrillæ of the part being irritated by the tetanin from the bacilli which are in the immediate neighbourhood, so that further amputation of the tail or excision of the nerve will afford relief. The wounds, or the seats of them, if healed, are in all cases to be fomented, and after being thoroughly cleansed, should be saturated with weak tincture of iodine twice daily. Should tetanus occur soon after an injury is inflicted, the wound should be examined, and any lacerated or partially divided nerve, foreign body, or dead tissue removed.

There are some cases of tetanus so acute from their commencement that it is quite hopeless to expect any but a fatal termination; and in every case where all the symptoms are firmly established before the fourth day of attack, death may be expected. But in cases where the symptoms are slowly developed, some movement of the jaws still remaining, the exacerbations not very severe—more especially if the animal possess a calm quiet temper, and lives over the ninth day—a recovery may be expected.

The wound having been properly attended to, the next thing to be done is to place the animal loosely in slings; this ought to be done early, and before the nervous excitability becomes too great. If the patient is comfortably slung, he will get used to the slings before the malady has attained its height. I recommend the slings, because many horses which are in a fair way of recovery lie or fall down when the muscles begin

to relax, and, when down, struggle and fight to such an extent that they seldom recover from the excitement and renewed severity of the disease thus brought on. The surroundings of the patient are of the utmost importance; the stable must be darkened; should contain no other horses; be situated in a quiet spot, removed from noises, and the door must have a lock, a key of which is to be kept by one individual (the veterinary surgeon, if possible), who is to visit the patient at most twice a day, and great care must be taken that the animal is not tormented by flies.

As a rule, the desire for food continues for several days; the thirst is considerable, and large quantities of nutritious fluids will be drunk with avidity.

Quietude having been secured, nourishment is to be given by allowing the patient milk and thick gruel to drink; along with these, eggs may be mixed with advantage. A little hay or grass placed in the rack will often keep the animal quiet, although the attempts to swallow sometimes cause a paroxysm.

The medicinal agents that have been used in the treatment of tetanus are numerous—purgatives, opium, tobacco, Calabar bean, woorara, prussic acid, calomel, chloroform, belladonna, hyoscyamus, cannabis indica, arsenic, chloral-hydrate, &c., &c. I have treated tetanus in various ways, and am satisfied that administration of a dose of aloes, if it can be given without exciting the horse, followed by belladonna—which is only to be given when the patient shows symptoms of great excitement—is the best method of treatment. The Calabar bean, given in doses of two to four ounces of the tincture, has a most wonderful effect upon the spasms, the pulse, and the breathing; but this effect is very transient, and is succeeded by a return of the spasms with great severity. The seat of the wound is from time to time to be smeared with the extract of belladonna; and when the belladonna is administered internally, it should be either dissolved in the animal's mash or drink, or else placed between his teeth, allowance being made for the probable waste.

The prussic acid treatment, so highly recommended by the late Mr. Lawson of Manchester, has with me proved to have no special superiority; and doubtless the success of Mr.

Lawson in the treatment of tetanus was due more to the tact and skill of the man than to any virtue contained in the remedy.

The reviser of this work is greatly in favour of the internal administration of iodide of potassium and the external application of weak tincture of iodine twice daily, along the course of the nerves of the injured limb, and also to the wound itself.

*Anti-tetantin* at one time was considered of considerable value in the *treatment* of the disease, but it has not proved successful; although as a preventive of the disease, if injected in doses of 10 to 20 c.c. for the horse, within a day or so of an accident, or at the time of a surgical operation, it is of the utmost value, and should always be made use of by veterinary surgeons whenever they perform operations or have wounded patients.

During an experience abroad, acting as Veterinary Adviser to the British Remount Commission, some tens of thousands of horses, cobs, and mules (all of which came long distances by train) were received by me for shipment across the seas. Many on arrival were found to have severe punctured wounds, either from gathered nails, or running into stakes, or by the prods of the nigger's driving-pole, often with a nail in the end of it.

I had every injured animal injected with anti-tetantin, and had not one single case of tetanus, though the disease was rampant in that country, which was very swampy, and the local practitioners could always show me a case or two.


Working in the knowledge that the toxin of tetanus combines specially with nerve-matter—in fact, seems to have an affinity for it—Fiebiger takes the fresh brain of a lamb, emulsifies it with physiological salt solution, and injects about 16 ounces of it below the skin.

This seems to be a good curative agent in many cases of tetanus, and is certainly well worth trying, as anti-tetantin seems of little or no value once the disease is established.

Those cases of tetanus which terminate favourably take usually about six weeks before the spasmodic contractions entirely subside. As soon as they can eat good food, they are to have it liberally. Corn, roots, and hay in the winter,

corn and grass in the summer, and a few doses of tonic medicine, such as the sulphate of iron, will materially assist convalescence.

In the fatal cases of tetanus, the breath very frequently becomes foetid prior to death, and if the mouth be examined, a quantity of slate-coloured epithelium will be found on the inner surfaces of the lips, gums, and tongue.





## CHAPTER XXXVII.

### CONTAGIOUS DISEASES—*continued.*

#### HÆMORRHAGIC SEPTICÆMIA (OR PASTEURELLOSIS).

ACCORDING to some authorities, this hæmorrhagic septicæmia is a most inclusive term, and is applied to a variety of diseases due to *Pasteurella*—namely, *deer disease*, *buffalo disease*, *gloss anthrax*, *swine plague*, *fowl cholera*, *fowl diphtheria*, *contagious pneumonia of the horse*, *Irish white scour in calves*, *influenza in horses*, *distemper in dogs*, *Stuttgart's disease in dogs*, *septic pleuro-pneumonia of calves*, *cornstalk disease of cattle*, and *Newmarket fever*.

In all of these diseases the causal agent is a cocco-bacillus, non-motile, polymorphous, both aërobic and anaërobic, and which does not form spores nor does it stain by Gram's method. These cocco-bacilli, or *Pasteurella*, appear to be everywhere—in water, soil, food, in the respiratory and digestive tracts of all animals, and seem as if lying in wait for an opportunity to gain access to the animal economy and give rise to disease. In cattle we frequently see three forms of the disease :

1. *The exanthematic form*, which is characterised by its rapidity of onset and course and its fatal termination. The animal ceases to feed or ruminate, the temperature may rise as high as 108° to 109° F., the breathing becomes hurried and laboured, and there is frequently a bloody discharge from the nostrils and bowels, large swellings of a tense nature appear at the throat, in the dewlap, the fetlocks, and behind the shoulders.

In many cases there is so much swelling round the throat that the tongue becomes purple and swollen and protrudes

from the mouth, and there is interference both with deglutition and respiration, and the animals rapidly succumb.

2. *The lung form*, in which there are exanthematic lesions with a croupous pneumonia and a pleurisy, the exudate into the chest cavity being very large in quantity.

3. *The intestinal form*, in which there are gastritis and enteritis, and in these cases there are usually the skin and subcutaneous tissue exudates.

On *post mortem* examination, the most marked lesions are exudations of serum into subcutaneous tissues and around the various lymphatic glands, and a general dissemination of minute hæmorrhages or petechiæ throughout the subserous and submucous membranes.

Microscopic examination reveals the presence of the organism, and subcutaneous injection of infected fluid into horses, cattle, pigs, or rabbits, causes death, with similar symptoms, in about twenty-four to forty-eight hours.

The disease is often enzootic, and is found on marshy, low-lying pastures and in badly-drained and ill-ventilated stables.

Preventive measures to be adopted are: Remove the animals from unhealthy surroundings, administer hypsulphite of soda night and morning, and build up the strength with good foods and tonics.

If necessary, the unaffected animals may be protected by injecting what is known as Lignière's polyvalent vaccine.

This vaccine consists of a mixture of the cultures of the various *Pasteurellæ*, and is used in two strengths—firstly, an injection of a weak vaccine; and, secondly, a stronger vaccine in the course of about twelve or fifteen days.

This protection is only expected to last about a year.

It is to be noted, in many cases of disease and death, occurring in live stock, particularly in cattle, due to the drinking of water contaminated with decomposing animal and vegetable matter, that the actual cause of death is due to a *Pasteurella* invasion. The animal's system becomes so depressed and vitiated that it can no longer resist the entrance of the *Pasteurella*, and thus we have various forms of hæmorrhagic septicæmia, some even resembling rinderpest, others akin to anthrax, and others, again, exhibiting a septic condition of the womb.

Bowhill, a well-known veterinary pathologist and a most careful observer, says in his 1906 *Report to the Department of Agriculture, Cape of Good Hope* :

"I am now in a position to present for your further consideration and information experimental proof in support of my previous contentions regarding the rôle played by organisms of the *Pasteurella* and allied group of bacteria in some of the prevailing diseases in the Eastern Coastal Districts. From the various animals I have been able to isolate typical cultures of the organisms belonging to the *Pasteurella* and allied species, and in most instances have been able to produce a similar disease with cultures obtained from the original species. The disease amongst the cattle, especially the acute cases, have in many instances been mistaken for anthrax. The organisms isolated from the various animals are morphologically indistinguishable one from the other, but this does not prove that they are one and the same. They are, however, individually capable of causing a specific disease under natural conditions in animals specially predisposed to their action. Nevertheless, some of the organisms under consideration have produced death, associated with specific lesions, in more than one species of these animals, when experimentally inoculated. It has been inferred that these organisms have probably a common origin, but that some of them are one and the same has not yet been definitely proved. The results included in this report tend to prove the existence of a very close relationship between the organisms associated with *Pasteurella* diseases in cattle and sheep. According to Lignière, many of the organisms of the *Pasteurella* group possess the important faculty of causing ephemeral infections 'so mild that the disease often passes unobserved. Although no symptoms become apparent, the toxins produced facilitate a secondary, multiple, or varied (sometimes) terminal infection by organisms present in either the alimentary or respiratory tracts.' Lignière's observations have enabled the exact nature of many diseases to be determined which were heretofore imperfectly understood.

#### "PASTEURELLOSIS OF ADULT CATTLE.

"The prevalence of this form of *Pasteurella* infection in the coast-lying districts is a very serious matter, as the out-

breaks are becoming more frequent. It has recently been reported on farms where it was previously unknown. There is also evidence that the non-burial or burning of the carcasses of animals dead from this malady harbours infection as well as being the means of the disease being spread to neighbouring farms by natives, wild dogs, jackals, &c. Another source of infection is by means of water contaminated with the dung and urine of infected animals. In the introductory portion of this report it is stated that organisms similar to those described by Lignière as belonging to the *Pasteurella* group were isolated from the lesions found in cattle dead of this infection. There is also another very important point to be considered in connection with the cultures obtained, and that is the symbiosis or association of other organisms with those of the *Pasteurella* group observed. This, no doubt, plays a very important rôle in the course of this disease, and an undoubted active part in many virulent and rapidly fatal cases. I have repeatedly found the *Bacillus coli*, *Proteus vulgaris*, also *Staphylococci* and *Streptococci* associated with the *Pasteurella* organisms. In many instances, it was only after carefully plating the growths that I was able to obtain pure *Pasteurella* cultures, the pleomorphic characters of these bacteria causing a lot of extra plating. It must be noted that animals of the same species exhibit a varied susceptibility to the same organism under similar conditions; this is increased by bad or insufficient food or exposure, which all tend to lower animal vitality. In many instances the disease appears in an acute form; at other times it runs a chronic lingering course; while in others it remains apparently latent, and when the natural resistance of the system is lowered by debilitating natural causes or inoculations with other organisms, vaccines, &c., the dormant *Pasteurella* organisms suddenly acquire an active rôle, resulting in a terminal infection. It is in cases of this nature that a mixed growth develops when cultures are instituted. It is also in such cases that the true nature of the *Pasteurella* infection is often overlooked, and the animal's death assigned to some other cause. Sanarelli, when he was experimenting with *Bacillus typhi abdominalis* of man, found that his inoculation experiments with the experimental animals were rendered much more effectual

when the animals were first inoculated with products of the growth of *Proteus vulgaris*, *Bacillus prodigiosus*, and *Bacillus coli*.

"I maintain that a similar condition exists amongst animals acquiring *Pasteurella* infections on this coast. If the intestinal canal be healthy and free from abrasions, ulcers, &c., and there is no abnormal acquirement of the *Bacillus coli* or other organisms by contaminated drinking-water, &c., there will be consequently no favourable ground for the multiplication of the *Pasteurella* organisms. The localisation of the lesions, the low adynamic fever, the foul odour of the ejections, and the terminal complications in many of the chronic cases of *Pasteurella bovis* bear a striking resemblance to paratyphoid in man. In the attenuated form, when the lesions are more or less limited in their extent, it appears probable that in the chronic cases the prolongation of the fatal issue, and consequent continuous production of toxin, accounts for the great anæmia present in both cattle and sheep *Pasteurelloses*; also the microbic changes in the various organs, especially the liver and the abdominal lymph-glands, as well as the derangement of the nervous system, whereby the disease is often erroneously described as *Lamziekte*, a bone disease stated to be due to the want of phosphates. It is a noteworthy fact that bones taken from typical cases were found on analysis to be normal in the chemical constituents specially described as wanting in *Lamziekte*. It has lately been shown that the reaction of bone-marrow to septic infection is a very delicate one. There is very early a congestion of the vessels, and increase of the leucoblastic elements, and a disappearance of the fat cells. Carnegie Dixon, in an Edinburgh University thesis (stated as not yet published), shows that the congestion appeared in two to three hours after infection, and leucoblastic transformation is well marked in twenty-four to forty-eight hours.

"This will no doubt explain why the congested state of the bones in many of these cases has led to a misunderstanding of the true nature of the disease. In the lymph-glands examined, necrosis of the lymphatic or adenoid tissue and hæmorrhage into the medullary spaces were observed, the organism evidently causing a bacterial infarction of the

vessels of the cortex and medulla. This agglomeration or clumping of the organisms no doubt enables them to increase and elaborate toxins. The above changes account for the many involution forms observed in smears prepared from these organs, as well as the difficulty of obtaining cultures in many chronic cases, a difficulty not anticipated when the prepared smears were found on microscopical examination to be teeming with bacteria. In 1904 I described three forms of this bovine infection as prevalent in the coastal area, and since then I have not found any reason to alter the opinion I expressed at that time. The form associated with a swelling of the throat is very fatal. An œdema of the neck, head, and tongue develops in a few hours, associated with great difficulty in breathing, frothing at the mouth, the animal being totally unable to swallow, and, owing to pressure on the larynx, the breathing is very noisy. The visible mucous membranes are cyanotic, and the tongue, being very much swollen, hangs out. Death occurs sometimes in a few hours, and sometimes the animal dies from suffocation about the third or fourth day. Diarrhœa is often present, and at other times the dung is dark coloured, hard, and covered with large pieces of bloody mucus, the odour being most offensive. There is another form that has been of very frequent occurrence this year. It is associated with extensive thoracic lesions, the chest being full of exudate and also the pericardial sac, the lungs being hyperæmic and œdematous, the same being well-marked in the interlobular septa of the lung."

## CHAPTER XXXVIII.

### CONTAGIOUS DISEASES—*continued.*

#### CROUP.

YOUNG cattle, varying from a few weeks to a few months old, are subject, when kept on low, damp pastures, more especially in meadows near rivers, and during the fall of the year, to a form of inflammation of the throat, characterised by the formation of a fibrinous exudate or false membrane of a greyish-white colour, sometimes brown or yellow, extending over the larynx, trachea, and sometimes fauces, or the bronchial mucous membrane.

Croup differs from ordinary catarrhal laryngitis in a most remarkable manner. In laryngitis there is an increased formation of mucus, which is discharged as quickly as it is formed. In croup, an exudative process attends the inflammation in the larynx and trachea, which induces the formation of false membranes, varying in thickness and consistency, some of which being several lines in thickness and very opaque, whilst others are so thin that the mucous membrane is easily seen through them. Some are so firm in consistence that they can be detached for a considerable length without tearing, whilst others are almost diffuent. In colour they vary from a dirty greyish-white to a yellow or yellowish-brown. If an animal be examined *post mortem*, after the disease has existed for four or five days, the windpipe and larynx will be lined for a considerable distance, in some places partially only; in other parts the entire circumference will be embraced by the false membrane, forming a complete hollow tube or cylinder.

The cause of the disease is the necrosis bacillus, a long, slender, thread-like organism, which is Gram negative, and so difficult to stain in tissue.

*Symptoms.*—These generally commence with a hoarse cough, discharge of frothy saliva from the mouth, and of mucus from the nose; the animal is unthrifty, has some difficulty in swallowing; sometimes swellings appear in the parotid and submaxillary regions, succeeded by difficulty in breathing, the inspiratory act being accompanied by a crowing noise, and by spasm of the laryngeal muscles, causing violent paroxysms. In other cases the spasmodic affection of the larynx and difficult breathing may occur without any premonitory warning. The pulse, hard and quick at the commencement, becomes feeble and indistinct as the disease advances, the fits of coughing more and more troublesome and violent; paroxysms constantly occur, particularly if the animal be subjected to any sudden excitement. In the course of two or three days flakes of the grey non-vascular and unorganised false membrane are coughed up; the expectoration becomes more profuse; being loosened and detached by a fluid poured from the mucous follicles, until finally separated and cast out; and if the case progresses favourably the false membrane is not again formed, the suppurative process terminating the inflammatory action.

If the stethoscope be applied to the larynx and trachea, a peculiar trembling may be detected in places where false membrane exists.

*Treatment.*—When the symptoms are very alarming, the breathing difficult, and the noise loud, tracheotomy should be immediately performed; indeed, it is essential that air be admitted early in every severe case, for should the operation be delayed, the blood becomes so overloaded with carbonic acid and effete materials, that the animal succumbs to a condition of blood poisoning.

The head is to be steamed for several hours consecutively, and the hot water used for that purpose may contain carbolic acid or iodine. Nitrate of silver in solution may be directly applied to the diseased mucous membrane, as directed in a former page. The solution commonly in use contains half a drachm to the ounce of distilled water.

The medicinal treatment will greatly depend upon the condition of the animal, but little good need be expected from any very heroic remedies. In the early stages the nitrate or chlorate



of potash are to be given in small doses, or the hyposulphite of soda in the animal's drinking water. If there be much prostration, spirits of nitrous ether, with camphor, may be prescribed, and in all cases a gentle oleaginous purge may be ordered if the bowels are costive. It must not be forgotten that the patient must be kept in a warm, dry shed, and have the body clothed if the weather be cold. The croup of fowls must not be confused with this croup of calves, as it is usually parasitic and due to the thread-worm, *Syngamus* (*vide* Parasites).

## DIPHTHERIA.

Diphtheria may be described as a specific blood disease, associated with sore throat of great severity, attended with extreme prostration, and characterised by exudation of false membrane upon and pulpification of the mucous membrane of the throat and sometimes that of the nose.

In the *Veterinary Journal* for August, 1875, Mr. W. Robertson, Kelso, reports some facts in connection with the occurrence of diphtheria in dogs and horses, and places on record the causes, spread, and subsidences in two particular outbreaks—one amongst horses, the other amongst dogs.

Mr. Robertson restricts the term "diphtheria" to "that specific sore throat ordinarily regarded as contagious, accompanied with much systemic disturbance, and tending to laryngeal croup." The difference between a croupous and diphtheritic membrane is that in the former the false membrane may be stripped off, leaving, under favourable conditions, a healthy granulous surface; in the latter this membrane is adherent and will not strip off, but tends to increase in thickness and plasticity. The disease diphtheria does not exist in the lower animals, except possibly in the cat; but there are conditions in which there is the formation of a diphtheritic membrane, although it is unassociated with Löffler's bacillus of diphtheria.

In both the outbreaks the fatality was great, all the horses—five in number—dying; in the case of the dogs, there were three or four recoveries from between thirty and forty seizures.

As seen in the horses the seizure was in every instance sudden; the animals—farm horses in good working condition, of different

ages--apparently in the enjoyment of the fullest amount of health and vigour one day, were on the following found unaccountably ill; two, indeed, were only noticed unwell in the evening, having worked all day, and fed with their usual zest, both in the morning and at mid-day. Attention was first directed to the animals from their inability or disinclination to drink when offered water; some pushed their noses into the trough or pail, and seemed attempting to swallow, but at once desisted, or the water returned by the nose. The breathing was at the same time noticed to be rather faster than natural, and the nose slightly pushed forward; occasional muscular tremors at this time showed themselves, and the animals were rather restless or uneasy. There was little or no cough, and when it did exist, it seemed merely to result from attempts made to swallow. The glands of the throat were slightly swollen from the first, but certainly did not increase much during the period the animals lived. The temperature rose rapidly, and continued high until shortly before death; while the pulse was accelerated about one-half, but was considerably less in volume and force. In one case there were well-marked symptoms of abdominal pain from the outset of the disease, with no abdominal organic lesion observable after death to account for these. In this instance there was also very noisy breathing; this, however, was accounted for at the *post mortem* examination, which showed more extensive involvement of the larynx and trachea than any of the others. None of these animals lived over the fifth day, and two of them died within forty-eight hours.

I have never witnessed a disease similar to that described by Mr. Robertson in the horse; but during the winter 1878-9 I had the opportunity of seeing several dogs, the property of various owners, that had this fatal form of sore throat, and which presented signs very nearly approaching those of diphtheria as described by physicians.

The symptoms were those of great prostration and languor; flow of tears from the eyes; a very sunken and pinched appearance of the face; the power of swallowing completely lost; inability to close the mouth, which in all cases was persistently open, the muscles of mastication, as well as those of deglutition, having lost their function either from paralysis or inflammation. Indeed, the first symptom observed was a dropping of the lower

jaw, and on this account it was supposed by the owners the disease was dumb madness; a more or less copious discharge of a viscid ropy saliva from the mouth, dirty yellowish-red tongue, the neck stretched and rigid, the glands slightly swollen, cedematous, and painful to the touch. Diarrhœa was present in all, and the posterior extremities were paralyzed.

The mucous membrane of the mouth and fauces was of a dark red colour, swollen, tense, and glistening from extraneous infiltration, but no ulcers were observable.

Three of the dogs died in from twenty-four to forty hours after they were first observed, but the fourth, a staghound, lived for four days, the symptoms in it having been much more gradually developed. In none of the dogs were convulsions or coma present; they all remained conscious to the last, evidently dying from diarrhœa and exhaustion.

No history could be obtained with any of the dogs. One was a lady's pet dog, and was daintily fed; another was a brewer's yard dog, always on the chain; so it was impossible, in these two cases at least, that the disease could have been induced by any tainted—infected—food, even if diphtheria had prevailed amongst the inhabitants of Edinburgh. Such, however, was not the case, diphtheria being entirely absent at that time.

The disease did not seem contagious or infectious, there being several dogs at the College at the time; some of these, owned by students, being confined in the boxes in which the diseased ones had died for the purpose of testing its contagiousness. One dog was inoculated, the results being negative.

Mr. Robertson traces the outbreaks which he witnessed to unsatisfactory sanitary conditions of the stable and kennels. These being drained and better ventilated the disease disappeared.

*Post mortem appearances.*—The *post mortem* appearances in four different cases examined at the College in 1879 were pretty much alike. A frothy mucus escaped from the nose and mouth, and the joints remained quite flaccid for three or four days.

On cutting through the skin, the larger veins were found to be engorged with extremely black fluid blood; and on pricking an artery, blood of the same colour and consistency exuded from it. The mucous membrane of the mouth, and all other mucous cavities, was very much congested, and covered by a frothy mucus; that of the fauces and tonsillar cavities being extremely

so in two cases, having commenced to undergo granular degeneration; in a third, a false membrane had formed over the glottis.

All the cavities of the heart were filled with black fluid blood, and only here was there any appearance of its coagulating, and the pericardial sac contained a quantity of fluid.

The surface of the lungs looked unnaturally red, with here and there small dark spots; and on cutting into its tissue, the same dark fluid blood exuded from it.

The stomachs, as in rabies, in two cases were filled with straw, stones, and other rubbish; but in the other two no such materials were found there.

The kidneys were also engorged with black blood.

Some interesting experiments are published in the *Veterinary Journal* for August 1875 as to the transmissibility of diphtheria, which the reader may profitably consult.

*Treatment.*—The treatment indicated by the symptoms is that calculated to destroy a virus, and for this purpose antiseptics, such as carbolic acid, might be worthy of trial. If the power of swallowing be completely lost, subcutaneous injections might be tried, and the throat repeatedly dressed with it or a solution of permanganate of potash.

Experimental inoculations with the diphtheric products of man have hitherto failed to induce the disease in animals, but it has been discovered that the serum of horses inoculated with diphtheric products furnishes an antitoxin which not only gives immunity, but has a powerful curative action when the disease is actually established in the human being; if applied in the early stages, the mortality is reduced as much as two-thirds.

## CHAPTER XXXIX.

### ENZOOTIC AND EPIZOOTIC DISEASES.

#### INFLUENZA.

UNDER this term is included three diseases, which every now and then prevail as epizootics in this and other countries, and which have been indiscriminately denominated *fever of the horse*, *epizootic of the horse*, *nervous fever*, *putrid fever*, *infectious pneumonia*, *typhus*, &c.

Falke, however, has reduced these to two conditions recognised under the term *influenza*—namely, the *red disease*, for which he has reserved the term *influenza*, and the other form he has named *typhus*. Whilst giving due credit to Falke for endeavouring to abolish the mystification concerning *influenza*, I feel constrained to state that under the term I can recognise three different morbid conditions, namely—(1) *Epizootic catarrhal fever*; (2) *Epizootic cellulitis* or *pink-eye*; and (3), *Epizootic pneumonia*, all being due to sapræmic organisms generally existent in large horse establishments, and liable to cause epizootic outbreaks under certain alterations of external surroundings, which seem to determine either the receptivity of animals subjected to their action, or a transformation of the vital properties of the microbes. For example, the disease may have been absent in a stable for a period of time, or until fresh animals are introduced. Then there is an outbreak of the malady amongst the freshly-introduced animals, and from which it spreads indefinitely amongst those which have had immediate or mediate contact. Here I think we have an example of the action of a non-contagious facultative parasite becoming transformed during its passage through the animal body into a contagious parasitic microbe, and which at first is thus increased in virulence, and after a period of time the

intensity of this virulence seems to become exhausted, and the microbe again is transformed to its original nature, often to remain dormant until some cause is brought into operation by which its morbid effects are again reproduced.

1st.—PANZOOTIC CATARRHAL FEVER OR INFLUENZA.

An epizootic febrile disease attended with great prostration of strength, and with early inflammation of the nasal, laryngeal, and sometimes bronchial mucous membrane, complicated with irritability of the digestive mucous membrane. Occasionally the disease implicates the substance of the lungs, pleura, liver, the fibrous structures of the articulations, thecæ of muscles and tendons, and the connective tissue of various parts of the body.

*Synonyms.*—Distemper; epidemic catarrh; epizootic catarrhal fever; (F.) *courbature*, *morfondure*; (L.) febris catarrhalis, defluxio catarrhalis, &c. The disease was first called influenza in Italy in the seventeenth century, because it was attributed to the *influence* of the stars.

Panzootic catarrhal fever or influenza has a very early history, but to trace this would be beyond the purpose of this work. In 1299 it appeared in Seville, and is referred to by the veterinarians Martin Arrendondo and Fernando Calvo, who derived their information from Laurentius Rusius. It killed more than one thousand horses and seemed to be incurable.

"In 1648 an epizootic broke out amongst the horses of the French army in Germany, and is described by Solleysell. It began by fever, great prostration, and tears running from the eyes, and there was an abundant discharge of a greenish colour from the nostrils."—(FLEMING.) In 1688 influenza was prevalent over the whole of Europe, affecting both men and horses. In 1699 Europe and America suffered from the disease, and again in 1732 it prevailed in both hemispheres. In 1767 it once more appeared in both hemispheres, and it is recorded that both horses and dogs were liable to its attacks. In 1776, after a very severe winter and warm summer, with an earthquake in Wales, influenza spread over Europe, attacking horses and dogs first, and human beings after. Poultry died in great numbers of an epizootic with defluxion from the eyes. This epizootic was observed in Edinburgh in December, and in England at the commencement of January

1788. Influenza was very severe in New York, and caused great mortality amongst the horses of Maryland. During the present century the disease has raged with varying degrees of virulence in 1849-50, 1863-4; and the last outbreaks we have to record are—

*Influenza in Britain in 1871-72.*—This outbreak was mostly confined to the English metropolis; it was of a severe but not fatal type; in 1891-92 it was general all over Great Britain.

*The American Horse Disease, 1872-73.*—This disease broke out in Toronto, Canada, on October 1st, 1872. In nine days it had attacked nearly all the horses in the city, and carriages could not be had for any price. On October 18th it had reached Montreal, and was prevalent throughout Canada. On 14th October it had reached Buffalo; 17th, Rochester; 22d, Boston, New York, Brooklyn, and Jersey City; 27th, Philadelphia; 28th, Washington. It made its appearance in Nova Scotia on October 13th.

Mr. James Law warmly advocates the contagious origin of this epizootic. Mr. Greene, M.R.C.V.S., St. John's, N.B., records the following important fact:—"I was always under the impression that influenza was both contagious and infectious till the late outbreak; since then I have altered my views with regard to the contagion and infection of that disease. One among several facts which I could mention will bear me out in this question.

"During the month of July 1872 a horse had been put to grass on Partridge Island, in the Bay of Fundy. This island is three miles distant from this city. No other horse had been near the island from the date of his landing up to the time of the outbreak in St. John's, N.B., and on the 15th or 16th of October, which was only two or three days after the first case was reported in this city, the horse on the island was affected with the most violent form of the epizootic.

"Would not the morbid matter have become diluted to such an extent (after travelling three miles) as to be inert?"—(*Veterinarian*, April 1873.)

#### ETIOLOGY.

The microbe cause of influenza is not determined. The disease may occur spontaneously in various places, but its first source of origin cannot be indicated. It does not depend upon any known condition of the atmosphere, nor upon soil, seasons, or temperature. It prevails on every soil and geological for-

mation, sometimes more so in low-lying districts than on the hills. It may appear at all times of the year; perhaps it is more commonly seen in autumn, winter, and spring than in summer. Very often, however, its worst form is seen during the hot summer months. Influenza has often appeared in foggy weather, but outbreaks have occurred without such a coincidence, and consequently no weight can be attached to this circumstance.

Its spread is not influenced by the wind; sometimes it moves against it.

"The outbreak of 1766 and the American one of 1872 succeeded to two very similar climatic conditions. The winters had been exceedingly severe and the summers unusually hot, and earthquakes had been frequent."—(LAW.)

Similar climatic conditions have, however, not been followed by an outbreak of influenza; and as in the case of fogs, mildews, &c., the coincidence can only be looked upon as accidental.

Sudden changes of temperature appear to assist the development of the influenza poison, and exposure to cold predisposes the animal to the disease, but neither of these causes is sufficient of itself to produce it.

Ozone in undue quantity in the atmosphere has been supposed to be a cause of influenza. This is, however, highly improbable, as ozone, although an irritant to the mucous membrane of the nose when applied in large quantities, has no effect on the nervous system like the influenza poison.

Influenza, like contagious pneumonia, is due to an organism of the *Pasteurella* variety, in all probability identical or nearly related to that organism. The diseases said to be due to these organisms are now very considerable, and it is somewhat remarkable in what different ways very similar organisms manifest themselves. We have perhaps accepted the *Pasteurella* too freely, but evidence is sufficient to show that these organisms, so prevalent and widespread in nature, are a never-ending source of trouble, and at all times have the power of acting in an actively pathogenic manner. Until further evidence is forthcoming we must accept the *Pasteurella* as the causative organism of influenza (see Hæmorrhagic Septicæmia).

*Predisposing causes.*—Animals crowded together in damp, ill-ventilated, and otherwise unhealthy situations, are generally



the first to suffer from influenza. In them it commits its greatest havoc. Young horses are more predisposed than those of maturer years; still, the old suffer severely, and are often carried away. Sex has no influence. Neglect of every description, as well as bad food and over-work, by debilitating, render animals subject to severe and early attacks of disease. But no amount of care will exempt them from it, as it appears in the stable of the nobleman as well as that of the poorest carter. The latter, however, experiences it in its greatest intensity, and at a much earlier period than the former.

#### PATHOLOGY.

The morbid agent absorbed into the blood gives rise to febrile disturbance and depression of the nervous centres. The period of latency, between the reception of the poison and the first manifestation of symptoms, is short but uncertain. To the febrile symptoms succeed those of the specific effect of the poison upon the mucous membrane of the nose, eyes, throat, and respiratory tract, as well as the intestinal mucous membrane and its ramifications in the biliary tubes. In some cases, the genito-urinal mucous membrane may participate in the inflammation, and in the majority there is more or less sympathetic or actual irritation of all the mucous membranes of the animal body.

In the majority of instances the fever precedes the catarrhal symptoms; the rigors, increased frequency of pulse, and elevation of temperature to 103° or 104° Fahrenheit, being observable prior to the appearance of any localization of the disorder. In other instances the irritation of the mucous membrane has been the first appreciable sign.

In uncomplicated cases the fever begins to abate in from three to five days, leaving the animal weak and prostrate.

It cannot be said that the fever terminates in a critical discharge, as in the human being. All that can be observed is that, coincident with the abatement of the fever, the secretions of the body become natural or slightly increased; the critical sweat, diuresis, or diarrhoea so generally observed in the human being, are usually absent in the horse.

*Complications.*—The catarrhal form is the one to be regarded as the simple or uncomplicated; to this is added, in some instances, pulmonic, gastro-enteric, hepatic, and rheumatic com-

plications, peri- or endo-carditis, the formation of thrombi, and nervous symptoms, as convulsive fits and coma.

*Symptoms of the catarrhal form.*—Actual rigor may or may not precede the other symptoms; very possibly the rigor is not observed. There is a dry, staring coat, the legs, ears, and nose are cold, with redness and dryness of the Schneiderian membrane, and an elevation of temperature to 103-4-5-7° F. or higher. There is sneezing, a hacking cough, and shortly a defluxion, from one or both nostrils, of at first a thin, more or less acrid mucus. The eyes are heavy, conjunctivæ injected and sometimes yellow, and tears flow over the face. The symptoms increase in intensity for two or even three days. The discharge from the nose becomes flaky and more profuse. In some cases the conjunctiva of one or both eyes is actually inflamed, as marked by opacity and imperfect vision or actual blindness, and there is partial or entire loss of appetite. The cough now becomes deep, sonorous, painful, and paroxysmal, convulsing the whole body, occasioning impatience, stamping of the feet, and great distress. The temperature of the surface of the body and extremities is very variable, sometimes high, sometimes low, or one leg hot, three legs cold, and *vice versa*. The pulse is feeble and easily compressed, numbering generally from sixty to eighty beats per minute. The mouth is hot and clammy, and the desire for water is intensified. The head is generally depressed. The animal is made to move with difficulty, and when compelled, he does so in an unsteady manner, swaying from side to side as if partially paralyzed. This muscular debility is much increased where there is great soreness of the throat and inability to swallow food or drink.

*Soreness of the throat* is indicated by “quidding of the food” (that is to say, the food is chewed and ejected from the mouth), or by its return through the nostrils. In the latter case an attempt to swallow excites a violent fit of coughing, and the food imprisoned behind the *velum pendulum palati* is forced into the nasal chambers. It there tinges the mucus discharges with its own colour; and when the horse is fed on grass the nasal discharge is thus turned green—a cause of great fear to some people, who at once conclude that the green discharge indicates something mysterious.

*The conditions of the secretions.*—In the earlier stages the *feces*

are dry, hard, pellety, and often covered with mucus ; the urine high-coloured and scanty, sometimes tinged with bile, sometimes opaque, and sometimes of the consistence and appearance of linseed oil, containing albumen, or loaded with urea and hippuric acid.

In a period of time varying from three to five days the symptoms begin to abate in intensity. The discharge from the nose becomes thick, yellow, and profuse; the cough looser, moist, and not so paroxysmal; the pulse gradually falls in number and improves in tone; the action of the heart is less jerky; the appetite improves; the throat can now be handled without causing the animal to cough. Any swelling of the throat which might have been present gradually subsides, and strength is generally restored in from twelve to fifteen days.

The leading character of the disease consists of an inflammation of the naso-pharyngeal mucous membrane; it also gives to the secretory organs a tendency to participate in the disease. Thus it is not at all uncommon for the submaxillary and parotid glands to become inflamed, swollen, and even to suppurate. When the critical stage has passed, the debility is generally very extreme, and the loss of flesh most marked. Indeed, it is a common observation that a horse in which the fever has abated and convalescence commenced, "looks as if the flesh has melted off his bones."

Owing to the debility of the circulation, dropsical swellings appear on the belly, legs, and chest. These, however, are not to be regarded in any way as serious, for as the animal regains its strength they will rapidly disappear.

In many instances, more especially in those which have been neglected or maltreated in the earlier stages, bronchitis is apt to supervene, and its accession is indicated by the cough becoming more paroxysmal, and the difficulty of breathing (dyspnoea) at first quite disproportionate to the other physical signs; the flanks heave, and the nostrils are widely dilated; in many instances the horse is said to fight for breath. The pulse, at first perhaps no quicker than the respiratory movements, which may be as frequent as 60 or 70 per minute, becomes very rapid—90, 100, or more beats per minute.

The auscultatory sounds are, roughness of the inspiratory murmur, best heard at the lower part of the trachea, and im-

mediately behind the scapula, or the sibilant rale heard over the greater part of the chest. The first indicates bronchitis, involving the larger bronchi, the second inflammation of the smaller tubes.

The visible mucous membranes become livid, dark purple, or leaden in colour, the animal is semi-torpid from the action of non-oxidized blood on the brain and medulla oblongata. The discharge from the nose differs from that seen in the uncomplicated form, being scanty, and sometimes streaked with blood, whilst in other cases dark-coloured blood, extravasated from the engorged bronchial blood-vessels, will issue from the nostrils.

The pulse now falters, cold sweats appear on the body, and the animal sinks on the sixth, seventh, or eighth day.

The *bronchitis* or *broncho-pneumonia* of influenza is distinguished from other chest affections by the greater difficulty of breathing, by the respiratory movements being relatively more disturbed than the action of the heart, by the lividity of the mucous membranes, and by the semi-comatose condition of the affected animal.

The *pneumonia* of influenza is characterised by a soft, dull cough, by rapidity of the pulse, great coldness of the extremities and surface of the body, by crepitation on auscultation and dulness on percussion of the lower parts of the chest, the *post mortem* revealing desquamation and proliferation of the bronchial epithelium, the presence of catarrhal products in the tubes and vesicles, and by effusion of serosity, mostly confined to the lower portions of the lungs. In consequence of the asthenic nature of the inflammation there is a tendency to gangrene and disintegration of the lung tissue. This termination—a rare one it must be admitted—is expressed by fœtor of the breath, sweats over the body, a faltering and excessively feeble pulse, rapid emaciation, looseness of the hair of the mane and tail, rapid sinking, and death.

Panzyotic catarrhal fever is not very frequently complicated with pleuritis, although, during some seasons, the latter disease rages as an epizootic.

*Abdominal or enteric complications.*—In all catarrhal affections there is great irritability of the gastro-enteric mucous membrane, hence all teachers of experience warn their students to be careful in administering purgatives. Succeeding to the naso-laryn-

geal symptoms, those of abdominal pain will become manifest, the animal frequently looking at its flanks, alternately lying down and rising, kicking at its belly, and frequently passing small quantities of hardened fæces, thickly coated and mixed with mucus. There is great debility, and some degree of torpor or somnolence; in some instances the sphincter ani is relaxed, and the anus constantly open, the animal straining incessantly, and exposing the intestinal mucous membrane of a deep red colour. The conjunctivæ are yellow; the tongue is coated, dry, and shrunk; the animal is thirsty, and the urine is high-coloured and scanty. Great care must be taken in properly diagnosing this condition, for, if treated as ordinary colic, the result is almost sure to be fatal.

Towards the termination of influenza *rheumatic complications* often appear; in some instances pain and stiffness in the joints appear early in the catarrhal form. Generally, however, rheumatism manifests itself towards the termination of the other symptoms. The animal becomes at first restless and uneasy, lifting its feet alternately from the ground; the joints emit a crackling sound when moved, then swell, after which the pain subsides. In other instances the rheumatic inflammation appears in some particular muscle, or more frequently tendon, especially the great flexors of the feet. The swelling and inflammation are generally situated immediately below the knee or hock, involving the flexors—perforans and perforatus. The swelling is very great, hot, painful, hard, and causing severe lameness, which sometimes remains for a long time after the animal has regained its health in other respects.

Few horses suffer from more than one attack during one season; many cases relapse, but one attack in no degree protects against another at some future period.

*Prognosis.*—If properly treated influenza is rarely fatal; the old, bad constitutioned, and over-worked horses only succumbing.

#### TREATMENT.

The practitioner is always to bear in mind that influenza is a disease which runs a definite course, and that it is in no way cut short by any interference. The treatment must consequently be directed to relieve any distressing symptom, to allay irritability, and to support the strength.

Comfortable clothing to the body and extremities ; food, consisting of warm mash of bran, boiled oats, linseed, or barley, and an abundance of good cold water for the animal to drink, are always to be recommended. It is good practice to see that the water is so placed that the animal can get at it at its pleasure, for in many instances, if it is not thirsty, it will wash its mouth and cool its tongue, and feel grateful for the relief thus afforded. Inhalations of warm water vapour, continued for an hour at a time, afford much relief, frequently mature the nasal discharges, and relieve the cough. The throat may be fomented with hot water and stimulated with an embrocation consisting of ammonia and oil, or at once blistered with the cantharides ointment. Mustard applications are very commonly used. I must confess that I never use mustard, for it causes very much distress, without securing any marked abatement of the disease. If there be much prostration, doses of spirits of nitrous ether, or of the carbonate of ammonia, may be given in cold water two or three times a day ; if the debility is not marked, nitrate of potash or sod. hyposulphite in the enteric form, dissolved in the animal's drink or mixed with its mash. Purgatives are always to be avoided, and any constipation which may be present in the earlier stages of the disease is best relieved by enemas of warm water. Should diarrhoea spontaneously occur, it must not be checked, at least if not excessive. Any irregularity of the bowels should be overcome more by food than by medicines. If costive, a laxative diet is to be given ; if the reverse, it may be advisable to give dry food.

If the appetite does not return in the course of the third or fourth day, milk should be substituted for water, and if the animal likes it, it should be allowed to drink of it abundantly—say three or four gallons per day. Skimmed milk suits better than unskimmed, as the latter is apt to induce some degree of diarrhoea. If at any time the milk disagree, and it will sometimes do so, it must be discontinued and gruel substituted. Neither milk nor gruel should be forced upon the animal by horning or bottling it down its throat in spite of its mute and often determined resistance ; and it is a point worthy of remembrance that food thus forced is only calculated to disorder the digestive organs, and destroy all chance of a return of appetite. In the human being, dog, &c., food thus forced would be

vomited, the stomach would at once be able to express its rebellion, but, in the horse, vomition rarely occurs; and who knows what unpleasantness or even pain is inflicted by thus disordering the already delicate digestion, by the common, but to my mind barbarous, practice of forcing sundry quarts of gruel, beer, &c., upon its unwilling stomach. Finally, never give the patient carrots, turnips, potatoes, or hard and raw food, till convalescence is well-established; if such should be done, there is every probability of a fatal termination from enteritis.

In mild attacks of influenza but little medical treatment is necessary; pure air, comfortable loose box, without draughts, well-drained and well-ventilated, with careful nursing and careful daily medical inspection, and the saline medicine above recommended, are all that is necessary. When convalescence has commenced, the restoration is materially assisted by a few doses of, first, vegetable, and, secondly, mineral tonics, good food, carefully regulated exercise, and careful grooming. If any cough remain, it may be necessary to apply a blister to the throat, and to administer such remedies as belladonna extract and camphor; and should the swelling of the limbs continue, the kidneys are to be stimulated by diuretics.

In some rare instances the local inflammation of the larynx is very great, and the tumefaction of the mucous membrane, vocal chords, &c., interferes with the act of respiration. When this occurs the horse's nose is "poked out," that is to say, the face is elevated, and the respiratory passages placed as nearly in a straight line as possible; the eyes become prominent, the inspiratory movement is performed with great difficulty, and accompanied by a loud, roaring sound. No time should be lost in giving relief to an animal in this state, and if fomentations and steaming fail to give relief, tracheotomy must be performed.—(See *Principles and Practice of Veterinary Surgery*, page 46.)

Influenza is also occasionally complicated with disorder of the liver, in which case the mucous membranes are more or less tinged with yellow; the bowels are constipated, or irregularly relaxed and torpid, the fæces are of a dirty clay colour, and foetid; the urine is high-coloured; the appetite is almost lost; and in some instances there are convulsive fits and comatose periods, which

cause much alarm. Professor Dick was of opinion that these cerebral symptoms were due to the extension of the nasal inflammation to the meninges of the brain, through the cribriform plates of the ethmoid. They are, however, due to the presence of biliary matters and products of degeneration of tissue in the blood, and are to be overcome by elimination through the natural channels. Some practitioners have termed this "bilious fever," but there is no necessity for any distinguishing term, as it is due to tumefaction of the lining membrane of the bile ducts preventing the free flow from the liver into the duodenum of bile, which, accumulating in the liver, is absorbed into the circulation, giving the visible mucous membranes the characteristic yellow, jaundiced tinge.

I have never found it necessary to administer any liver stimulant in this complication. All that is necessary is to keep the bowels regular by a mild aperient, such as a pint of linseed oil, and allow the animal such food as it will partake of which is of an easily digested and aperient nature. One condition which often prevails in this form requires modification, as it is apt to cause some degree of suffering, namely, an acid state of the stomach. The acidity of the stomach is manifested by grinding of the teeth, licking the walls, placing the tongue on cold objects, with drivelling of a clear saliva from the mouth. The bicarbonate of soda is very useful, giving speedy relief from this annoying symptom. The medicine is to be given two or three times a day, either dissolved in the drinking water, or as a draught out of a bottle.

If there be abdominal complications, the colicky pains are to be relieved by fomentations to the belly, and small doses of opium, the bowels being regulated by oil. But even here no very decided and heroic steps are to be taken to relieve symptoms, it being always kept in view that they are the result of a cause which will not expend itself until a certain period, and that mere remedies, although perhaps affording relief at the time, are generally debilitating in their effects, and often tend to cause a fatal termination.

The sequelæ of influenza are, hydrops pericardii, hydrothorax, ulceration of the larynx, roaring, and sometimes glanders and farcy.



## MORBID ANATOMY.

Uncomplicated influenza seldom proves fatal, except in the very old or otherwise debilitated animal; the most prominent lesions being great congestion of the mucous membrane of the respiratory track, extending even into the minute bronchi, some of which are filled with a muco-purulent discharge; the pulmonary lobules being consequently collapsed. The blood is dark, and is said to contain micrococci. The majority of fatal terminations are caused by the gravity of some local complication, such as pneumonia, bronchitis, and enteritis.

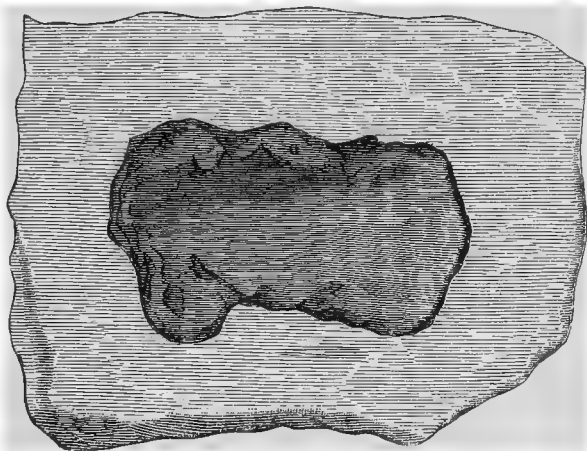


FIG. 35.—Congestive patch on mucous membrane of stomach.  
(Specimen sent by Mr. T. Taylor, V.S., Manchester.)

In Manchester it has been found that many dead animals present a dark patch of congestion, with incipient ulceration on the mucous membrane of the stomach, as shown above.

#### 2d.—EPIZOOTIC CELLULITIS, OR PINK-EYE.

The term "pink-eye" seems to be an importation from America—from one of the inconstant symptoms of the disease being redness of one or both eyes, and swelling of the eyelids.

Some suppose that it is a new disease, and that it first appeared in America in 1872, the actual seat of its outbreak being Toronto, Canada, where it appeared on October 1st, 1872; in nine days it had attacked nearly all the horses in the city,

and carriages could not be had at any price. On 18th October it had reached Montreal, and was prevalent throughout Canada. On October 14th it had reached Buffalo; 17th, Rochester; 22d, Boston, New York, Brooklyn, and Jersey City; 27th, Philadelphia; 28th, Washington; and had been witnessed in Nova Scotia as early as October 13th.

It is characterised by great and sudden prostration of strength, elevation of temperature and other signs of fever, with pain in and swelling of the extremities; generally redness of the conjunctivæ, swelling of the eyelids; and sometimes cranialgia; often colicky pains; congestion of the lungs, and in some rare instances an involvement of the brain and its membranes, expressed by phrenzy or coma.

The rate of mortality differs very widely,—from 1 to 35 per cent.

In fatal cases death is due to congestion and gangrene of the lungs, *ante mortem* clots in the heart and great vessels, enteritis, and extreme prostration of the system generally.

#### CAUSES.—ETIOLOGY.

Some writers maintain that it arises from contagion only, but there are many facts against this exclusive view. 1st. Its sudden and simultaneous occurrence in various parts of a district or city. For example, a place may be entirely free from the disease one day, and upon the next outbreaks will occur in many and widely separated spots in that place. 2d. Its occurrence in isolated places where contact with diseased animals is out of the question; for example, the first appearance of the disease in 1880 was at a farm in the Lothians at least one mile and a half from a railway station, and during the spring, when all the horses were kept at home ploughing. In a few days it was in Edinburgh and in many centres; but whilst I am convinced that it occurs as a malarial disease, I am equally convinced that it is highly contagious and infectious, and can be conveyed from diseased to healthy animals with great facility. Diëckerhoff says it may be readily communicated to healthy horses by the intravenous or subcutaneous injection of warm blood taken from a diseased animal; and Mr. Archibald Robinson, F.R.C.V.S., Greenock, says that it can be transmitted by the stallion which has had the disease months before to the mare by the act of coition.

A purely infectious disease like the pleuro-pneumonia of the ox follows the great lines of commerce, and can be traced from place to place with a certain degree of exactitude, but a miasmatic disease, which may afterwards become infectious or contagious, cannot be followed from place to place. I am therefore forced to the conclusion that, like Roman and typhoid fevers in man, this disease, arising as it does from the entrance of a microbe into the animal system, is not due so much to that germ itself, but to certain properties which it has obtained from perhaps unknown conditions of the air; that the germ itself is constant in the surroundings of animals and under ordinary conditions quite innocuous, but let those conditions be altered, the properties of the germ become virulent and infectious.

Now it appears that the infectious properties of this germ are generally developed during or immediately after a long continuance of wet weather. Several outbreaks within my memory have been so ushered, but this is not universal, as some writers state that it occurs in all weathers and in all climates. This might be more correctly said of the catarrhal form than of the other.

The microbes, which I have invariably discovered, as shown in the photo-micrographs, have a great tendency to arrange themselves in pairs. (See fig. 36.) They average about  $\frac{1}{24000}$  of an inch in size, grow freely in gelatine and agar-agar, and are easily stained with the aniline dyes. These organisms correspond to the description already given of a *Pasteurella* which is now considered as the causative organism, and is probably the same as that which causes influenza, being altered in its acute pathological effects by some altered conditions of environment.

The symptoms are as follows:—Some degree of lassitude; more or less failure of the appetite; the animal is inclined to be sluggish; his movements are clumsily performed, and he is inclined to “trip” with his toes; the eyes are heavy, eyelids partly closed, in many cases some increased redness and some swelling of eyelids, with a discharge of tears over the face, and sometimes opacity of the cornea, and fibrinous clots in the aqueous humour of the eye.

If examined at this stage, the temperature may be from 102° to 104°, or even 105°; the pulse from 60 to 70 per minute;

respirations not particularly hurried; and if the horse be stopped from all work at this period the severity of the disease may increase, but very slightly, and recovery may be expected in the course of a week or nine days; but if the animal be kept at work, or, even if not working, in a badly ventilated or otherwise improper stable, the symptoms rapidly increase in severity, the temperature rises to  $106^{\circ}$ ,  $107^{\circ}$ , or even as high as  $108^{\circ}$ , the respirations hurried, the pulse increased in frequency to 80 or even 110 beats per minute, is small and sometimes almost too indistinct to be felt by the most practised touch; the animal becomes excessively lame, sometimes immoveably so; or he is



FIG. 36.—Microbes of pink-eye, showing bipolar staining.

continually resting or pointing one foot and then the other, as if suffering agony in feet or limbs. In a short period of time the limbs begin to swell, more particularly about the lower joints, *i.e.*, the hocks and knees downwards, and as the swellings become pronounced the pain disappears.

As already stated, there is usually a cough and some degree of roughness of the throat and bronchial tubes, but in the last outbreak in Edinburgh these were conspicuous by their absence.

In some instances intestinal pain, expressed by restlessness, looking round at the flanks, scraping with the feet, and even

rolling about, is present shortly after the first manifestation of illness; and in others—and these are the really dangerously fatal ones—well-marked signs of inflammation of the bowels are observable at an early stage, with intermittent action of the heart and total loss of appetite, or even extreme abhorrence of food, and occasionally of water.

The bowels are at first normal or slightly constipated; the fæces covered with mucus (hence the terms typhus-mucosa and muco-enteritis), and their colour frequently indicates an absence of biliary secretion or the presence of altered bile; the rectum

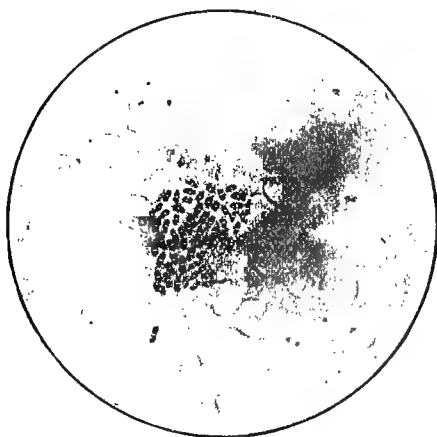


FIG. 37.—Microbes in groups.

is often irritable, and the passage of fæces causes some degree of pain; on the third or fourth day the fæces become pul-taceous and often of a brown colour, and later on diarrhoea may occur.

In other cases, including from the third to the sixth day of the disease, serious pneumonic symptoms are observed: there is an extensive exudation into the lung tissue—in fact, what is observable as taking place in the extremities, viz., exudation and swelling, is occurring in the lungs, causing the respiratory movements to become greatly accelerated, but if gangrene does not set in, indicated by fœtor of the breath or discharge of

dark-coloured blood from the nostrils, recovery may still be hoped for.

In other instances, even during convalescence, the pulse, hitherto moderately strong, and all other symptoms passing away, becomes rapidly feeble, and the animal suddenly dies, and on making a *post mortem* examination, firm clots or thrombi have been found in the heart, lungs, and blood-vessels, which have stopped the circulation and caused death.

This termination has to be very carefully guarded against, as it may occur in the most promising cases. It is brought about as follows:—The disease is one which particularly attacks areolar tissue; and inflammation of this tissue, like that of the more firmly fibrous, causes the development of coagulating properties in the blood, and when—added to this coagulative tendency—the heart loses its tone, the blood coagulates in its cavities and blocks the circulation. In other instances horses have died weeks or even months after they have passed through the disease, and *post mortem* examinations have revealed plugging of the vessels of some important organ.

Another rare complication is witnessed by symptoms of irritation of the brain and its membranes, with effusion into the ventricles and sub-arachnoid space. As this complication manifests itself there will be at first delirium, the eyes staring, the pupils dilated, perhaps complete blindness, with unconscious restlessness, tendency to elevate the head, followed by paralysis commencing in the hind limbs and extending to the whole muscular system, with total blindness, unconsciousness, stertorous breathing, inability to swallow, and loss of voluntary movements: these conditions indicate a rapidly approaching fatal termination. To recapitulate, the symptoms may be briefly stated to be as follows:—

Swelling of eyelids, often with diffuse redness of the conjunctivæ; congestion of the superficial vessels of the eyes, cloudiness of the cornea, and collection of yellowish fibrinous matter or blood-clots in the anterior chambers of the eyes. Sensitiveness to light, eyelids more or less closed, and discharge of tears; but in milder cases the eye symptoms may not be very distinct.

Pulse from 50 or 60 in mild to 100 or even 120 in severe cases.

Temperature from 103° or 104° in mild to 106°, 107°, or even 108° in severe cases. 108° was observed in one case only in the College practice, and ended in recovery. During convalescence the temperature rapidly falls, and in many cases to below the normal—to 99°, and rises to normal—about 101°.

Gait awkward and slow, loss of tone in the muscles, particularly those of limbs, ears, and lips, and sometimes marked weakness in the hind quarters, and sometimes very great lameness.

The respirations from 15 to 20 in mild to 30 or 40 in grave cases; these, however, are subject to modifications, as in some horses treated at the College we observed the respiration even slower than natural at first, and increasing in frequency during convalescence. In some fatal cases the breath is foetid for some time before death, and respirations very rapid.

Mucous membrane of the mouth of a venous red colour; tongue dry; fæces at first dry, but on third or fourth day brown and poultice-like in consistence; later on, diarrhoea in some cases.

Limbs slightly or considerably swollen; urine diminished, and sometimes albuminous, and in some cases of a chocolate colour, and containing broken-down blood-corpuscles.

#### POST MORTEM APPEARANCES.

*Heart.*—Congested; sometimes covered with mulberry coloured spots—petechiæ. Clots of blood in auricles, ventricles, and larger blood-vessels.

*Blood.*—Dark-coloured and viscid; white corpuscles increased in numbers, and many of the red ones broken down.

*Lungs.*—Generally congested; infiltrated with fluid, or filled with blood extravasation, which has undergone decomposition prior to death, and given rise to foetor of the breath.

*Liver — Kidneys.*—Generally congested. Kidneys sometimes black.

*Connective Tissue,* particularly of the limbs, contains much fluid or semi-coagulated exudate.

*Muscular System.*—Generally pale and flaccid.

*The Brain and its Membranes* congested, and the cranial cavities containing a superabundant quantity of fluid in those cases where frenzy or coma have appeared during life.

*Digestive System.*—Mucous membrane of stomach sometimes congested; bowels inflamed, and occasionally covered with petechial spots; contents generally fluid.

#### MORTALITY.

This seems to have varied during the 1890-91 outbreak in a most remarkable manner. Some practitioners who have replied to a circular issued by me, record a loss of only 1 to 2 per cent., whilst others return from 5 to 9; and it is reported that some have lost even a greater number. Our loss at the College was under 1 per cent., and at Piershill Barracks, out of a total of 59 treated by Mr. Phillips upon the principles of this College, there were no deaths.

#### TREATMENT.

The first and most important essential in the treatment of the disease is absolute rest as soon as the slightest indication of illness has been observed.

The animal, if possible, should be placed in a cool, well-ventilated, but not cold, loose box, warmly but not too heavily clothed with sheet and hood; and when bandages do not cause irritation the legs should be bandaged. The diet should consist of bran mashes, and a moderate quantity of hay for the first few days, except where a horse has been used to and is fond of a linseed mash. In such a case one pound of well-boiled linseed should be mixed with the mash every night until the bowels are loosened, when, if there be any signs of diarrhoea, it should be discontinued, and dry corn given. The mashes should be continued until the fever has subsided, and then gradually changed to the horse's ordinary diet. Cold water should be placed within the animal's reach, so that he can satisfy his, often inordinate, thirst night and day, and if greatly prostrate, the patient should be placed in slings. Medicinally, salines and other remedies recommended for the catarrhal form may be administered in accordance with the phase of the disease. If pain be prominent, the bromide of potassium has proved useful.



### 3d.—EPIZOOTIC PNEUMONIA, OR PLEURO-PNEUMONIA OF THE HORSE—LOBAR-PNEUMONIA.

*Definition.*—A croupous inflammation of the pleura and substance of the lungs, preceded and accompanied by a low typhoid or adynamic form of fever, which lasts from seven to fourteen days. It generally occurs but once in a season, but one attack does not render an animal exempt from a second or third. It is now considered an infectious disease, due to a micro-organism, which is asserted by some writers to be the *Diplococcus pneumonia* of Frankel, identical with that found in the saliva of certain persons, and which produces septicæmia in the rabbit. Others state that it is a streptococcus. Galtier and Violet describe two organisms—a streptococcus and diplococcus—whilst Cadéac states there is only one micrococcus, often grouped in pairs, sometimes in chains. The microbes of Galtier and Violet do not stain with Gram, whilst all the others do. They are all facultative anaërobcs.

The cause is undoubtedly a bipolar staining organism of the *Pasteurella* group which was at one time thought to be a diplococcus; but it is not, and has no relationship to the diplococcus of pneumonia of man. Some controversy on this subject has taken place on account of the frequency of streptococci in old cases of epizootic pneumonia, but this has already been explained in connection with *Pasteurella* infections. The original organisms, having disappeared, have given way for the appearance of others, whose presence is an accidental contamination. The pneumonia following on strangles, and due to the streptococci of Schultz, is an entirely different disease. It has been said that all pleurisies of the horse are due to streptococcus infection, and this may be so; but it does not follow, any more than that all pleurisies of man are due to tubercular infection. Such statements are too wide.

The epizootic pneumonia of the horse, which is rarely accompanied by a pleurisy, is most probably a complication of concurrent disease with influenza, and due to the same organism.

For the last few years this form of epizootic has prevailed to a very great extent both in the north and south of Britain. Erroneously called “influenza,” it has seemingly and for some

time taken the place of that affection, from which it differs very materially. The true uncomplicated influenza is a disease of the mucous structures: this affects the serous covering and substance of the lungs.

In 1861-2, also in 1891 and 1894, this form of epizootic disease became very prevalent in the north of England, where it raged for many months, committing great havoc amongst horses of all kinds, but particularly amongst those most exposed to the vicissitudes of the climate. It has prevailed more or less ever since, both in town and country, more particularly amongst young horses and those removed from pastures into stables. The knowledge that nearly every newly purchased horse is liable to suffer from this or some other form of epizootic disease, deters many persons from keeping horses, and hence we find that many whose business requires that horses should be employed, prefer to let out their work to contractors and carriers. This repugnance to purchase and keep horses by men in business has also been increased by the great mortality which has generally resulted from this epizootic.

#### SEMIOLOGY.

The primary symptoms are very often more or less obscure. The animal is dull, dejected, off its food, performs ordinary labour with difficulty; lassitude, perspiration, and fatigue are easily induced. Examined carefully, the pulse will number from sixty up to eighty per minute—during some seasons eighty has been the common number—and the animal temperature will vary from  $104^{\circ}$  to  $106^{\circ}$ . Sometimes there is a cough from the commencement; very often, however, there is no cough during the first three or four days. The extremities and ears are alternately hot and cold; the appetite is almost entirely lost; the alvine and urinary secretions are defective; the visible mucous membrane injected, and frequently of a rusty tinge; in some cases there is a rash in the mouth, a rusty or straw-coloured discharge from the nostrils. The animal does not lie down, Auscultation does not reveal much at first; in fact, for three or four days the animal may be said to be suffering from fever without local complications, but at the end of that period a hard, dry, painful cough is now and then heard; the thoracic walls are fixed; the breathing is abdominal; the elbows are

turned out, and a line or hollow extends from the ensiform cartilage of the sternum to the anterior spine of the ileum, which denotes that the ribs are fixed, owing to pain within the chest. If the horse is now made to move suddenly, it emits a grunt or groan; in some instances it groans at each expiration, and if slightly rapped in the chest with the knuckles it will do so with pain; the breathing is catching and short, and auscultation will detect a friction sound.—(See *Pleurisy*.) Very commonly the right side only is affected, in some cases the left, and in rare instances both sides.

In the course of the next ensuing two days, the sounds of the chest indicate pleurisy, pleuro-pneumonia, or a complication of these with pericarditis. From the commencement of the manifestation of chest disease, the breathing becomes somewhat hurried, the pulse assuming a hardness of character which it did not possess at first; the number of its beats are not further increased; on the contrary, rapidity of the pulse may decrease as the exudation on the pleural surfaces advances, and it is not at all uncommon for one to witness an increased rapidity of the respiratory movements, gradual condensation of lung tissue, some effusion into the thoracic cavity, and a diminution in the number of the pulsations. If pericardiac complications are present the pulse is marked by a peculiar indistinctiveness and irregularity; the cardiac beat is generally loud and short; the impulse, however, is weak, and the arterial pulsations indistinct.

In some instances the whole body becomes stiff and sore, and the horse is disinclined to move, but stands with its nose extended forwards, nostrils dilated, and fore legs apart, presenting an appearance of distress, which is heightened by frequent looks at its sides, and by attempts to lie down, as if suffering from colicky pains. In such instances the respiratory movements are short, shallow, and much accelerated, numbering fifty, sixty, or even eighty or more per minute, whereas in the majority of cases they do not number more than thirty per minute, when auscultation may detect considerable exudation into the lung tissue.

An examination of the respiratory apparatus by percussion and auscultation—the only satisfactory method of diagnosing chest diseases—will enable the practitioner to detect the various changes as they occur.

1. For the first two or three days after attack both percussion and auscultation often give negative results ; at the termination of that period percussion over the seat of the pleural inflammation will cause the animal to groan. Auscultation will detect a dry friction sound if the pleura be affected ; absence of true respiratory murmur, and the presence of crepitations, large or small, if the lung-substance be involved ; a combination of friction sound, and crepitations, if both pleura and lung tissue, and a "*to-and-fro*" rasping friction sound, with more or less palpitation, if the pericardium be involved.

These signs indicate—1*st*. That the natural pleural secretion is arrested, and that the dry pleural surfaces rubbing on each other cause the friction sound ; 2*d*. That engorgement of the lung tissue has taken place ; and 3*d*. That the pericardial secretion is arrested, causing the "*to-and-fro*" sound at the heart.

2. In the course of from one to two days the above abnormal sounds disappear. Auscultation now reveals that in the part affected there is no sound at all, or that a tubular or hollow sound is heard. The absence of sound indicates either that the lung tissue has become impervious to air, or that a considerable effusion of serum has occurred into the thoracic cavity. If the former, the absence of sound becomes apparent by degrees only ; that is to say, the respiratory sounds become gradually feeble as the consolidated part is approached, showing that at the border of the hepatized tissue the inflammatory process is less advanced. There may be crepitations or tubal sounds, but when the absence of sound is due to effusion of serum (hydrothorax) the sounds terminate abruptly at a certain distance from the floor of the thorax, the respiratory sounds being louder than natural above the line of termination, and in some instances the presence of fluid in the chest will be indicated by a "dripping sound," as of drops of water falling into a well. The tubal sounds indicate a less degree of consolidation ; that air finds ingress into some of the bronchial tubes, but not into the more minute ones and air cells.

This condition may exist for three or more days, and then the crepitations reappear, showing that the exudate is breaking up and becoming gradually removed by absorption ; the dull impervious part at the floor of the cavity becoming daily shallower, and the normal sounds slowly returning.

Perussion during the stages of consolidation and effusion produces a dull sound over the diseased part, and increased resonance over the healthy parts of the chest. In all the unaffected portions of the lungs the respiratory murmur is increased.

The "*to-and-fro*" cardiac friction sound also disappears, owing to effusion into the pericardial sac, and reappears as the exudate is removed by absorption.

Very often after convalescence has been established the cough becomes more frequent; it, however, loses its painful hacking character, being now louder and stronger. In some instances, more especially if the exudate has been very abundant, a mal-condition of the system is induced by the abundance of effete materials in the circulation, removed by absorption from the transformed exudates. This has been compared by Professor Gamgee to the hectic fever of the human being. There is much emaciation, imperfect digestion, capriciousness of the appetite; the hair is easily removed from the mane and tail; irregularity in the surface temperature; and continuance of the increased temperature of the body, as indicated by the thermometer. If this condition of the system be not successfully combated, the respirations again become accelerated and the ribs fixed; there is flapping of the nostrils, which are greatly distended; the animal again discontinues to lie down. Anasarca swellings may appear on the chest, abdomen, and the legs, denoting the presence of hydrothorax, to which the animal may eventually succumb in the course of an indefinite period—sometimes in two or three days only, sometimes in as many weeks—or symptoms of farcy or glanders may appear.

#### POST MORTEM APPEARANCES.

In very acute cases, the appearances revealed by a *post mortem* examination are, intense redness of the pleural surfaces of one or both sides of the chest; the engorgement of the vessels embracing the visceral and parietal surfaces, and some degree of effusion and exudation of lymph. In some instances, the lungs show but little disease, whilst in others patches of inflammation are found throughout their substance. The pericardium and endocardium are generally more or less injected, and the death of the animal at this early stage generally results more

from the cardiac complication than the pleural disease. It is but seldom that a case terminates so rapidly, unless the animal be kept at work after it has fallen ill.

The general symptoms of septicæmia, together with the hæmorrhages and heart symptoms, all tend to show that the disease is due to an organism of the *Pasteurella* variety.

If the horse live for two or three days after the pleural symptoms have become manifested, along with the injection of the vessels, an abundant quantity of serum will be effused into the pleural sac, floating in which, and loosely adherent to the pleural surfaces, bands of lymph will be found. These bands of lymph are loose and watery, and the serum is turbid or flocculent. If the horse has lain for some hours after death, and the *post mortem* is carefully performed, the effusion is clear at its upper and turbid at its lower part.

If the inflammatory process has been less rapid, the lymph is of a more consistent appearance, and forms over the inflamed parts flocculi of various sizes, or a distinct lining varying in thickness, and of a honey-combed appearance; but the lymph in the epizootic form does not possess the firmness, plasticity, and tendency to organization which is characteristic of that of the sporadic form of the disease. These bands of lymph are sometimes of very rapid formation, and they may even show some trace of organization in forty-eight hours after attack. This fact is of considerable importance legally, as it is often found that if a horse dies in a week or two after purchase, an attempt is made to compel the seller to refund the purchase-money on the ground that the animal was diseased prior to the date of sale. Old bands of lymph, and such as indicate previous disease, are firm, organized, of a glistening appearance, and unite the opposing surfaces more or less firmly together. New bands of lymph, on the contrary, are soft, watery, and the parts which they may attach together are easily separated.

In some instances, extravasations of blood give the lymph a red colour. This must not be mistaken for vascularity, as the latter would denote organization and age.

Examined microscopically, the lymph exhibits the presence of numerous inflammatory corpuscles, oil globules, and ill-developed fibres.

The exudate into the lung tissue is also wanting in plasticity,

the lung cuts up soft and moist, a whitish fluid oozing from the cut surfaces.

In some cases, where the vital powers have been very low, or where they have become weakened by injudicious treatment, an abundant, turbid, foetid fluid has been thrown out, partaking much of the character of unhealthy pus; or abscesses form in the lungs, and in rare instances gangrene of the lungs has occurred.

The pulmonary complication is generally found at the inferior portions of the lung, and in many instances the exudate indicates that the process is wanting in the true character of plastic inflammation.

#### TREATMENT.

From the foregoing observations, it will be understood that the local complications of this disease are preceded by fever, the pleural and pulmonary inflammation appearing after a few days have elapsed. The fever is of a typhoid or adynamic character, and the severity of the chest affection is generally in accordance with that of the premonitory fever. The fever, slight perhaps at first, is very often intensified by the suffering animal being kept at its ordinary work for some time after the appetite has failed. This inattention on the part of those in charge of horses is often a cause of great loss, inducing a fatal termination where recovery might reasonably have been expected. The febrile state and the local inflammation are increased by all debilitating influences, such as bad food, previous disease, natural delicacy of constitution, by the abstraction of blood, and very commonly by the administration of purgatives. Many horses are destroyed by the administration of aloes, a dose of which is often given when the horse is seen to be "off his feed." Superpurgation may not be induced, but the chest inflammation is much increased, and the animal often succumbs.

There is another practice in vogue, more particularly amongst horse-dealers, namely, that of giving a purgative (aloetic) ball to nearly every fresh horse they buy, and this is done without taking the health of the animal at the time into consideration at all. I am convinced that this is a most reprehensible practice,

leading to grave pulmonary diseases and to death, more especially if an epizootic is prevalent.

The pathology of chest inflammation leads us to the conclusion that when once established it runs through certain stages, namely, congestion of the vessels, the outpouring of an effusion, and the formation of an exudate. The congestion, effusion, and exudation are determined by the intensity of the preceding fever. If the fever is slight, the local disease will be slight also; if, on the contrary, the fever is severe, the local inflammation will be proportionately grave. During some seasons the disease from its commencement is severe, and then the weakly succumb. But at other times this is not the case, and very few deaths result from the disease; but its character is always rendered more serious by irrational treatment.

The duration of the fever cannot be cut short by any treatment, and attempts to do this are very often the cause of numerous deaths. Its severity, however, is amenable to modification by—

(1.) Complete rest whenever the slightest sign of illness is discoverable.

(2.) By housing the animal in a warm, dry, light, well-ventilated loose box.

(3.) By taking special precautions that no draught of cold wind blows upon it; for it must be remembered that cold, more especially cold wind, is the common cause of the disease, and that removal of the cause is the first step in the treatment of all diseases; and, again, that the severity of many affections is dependent on the “dose” of the cause. When this is heavy or long continued, the results will also be heavy and severe.

(4.) By clothing the animal and bandaging the legs, in fact by keeping up the equilibrium of the circulation; for if the vessels of the skin are prevented, by the operation of cold—acting upon and constricting them—from receiving a due supply of blood, internal congestions and inflammations are aggravated and often determined.

In addition to these rules, the practitioner must remember that the disease, once established, is the effect of a cause which has been in operation for some time past; that the effects, whatever they may be, are the natural physiological responses of the animal body to the action of such cause or causes; and that all attempts to remove such effects or conditions by other than those



processes which nature herself attempts, and generally accomplishes, can only result in disappointment and loss.

How, then, does the animal body rid itself of disease and its results? Briefly, in the one before us, the inflammation is the result of an irritant, acting for a certain time upon a certain tissue or organ. If the dose of the irritant is strong, the resulting inflammation will be strong also, and nothing will subdue that inflammation so long as the cause retains its strength and is allowed to operate. This inflammation is characterised by congestion, effusion, and exudation. When the cause is removed, or when its strength is exhausted, the congestion slowly disappears, and the products of the inflammation, namely (1.) the effusion, is gradually taken up into the circulation and removed from the body by the excretory organs; and (2.) the exudation or lymph, degenerated into a turbid fluid material—the pathological milk of Virchow—is removed in the same manner as the serous effusion, or is replaced by a fibrous structure, which binds the surface of the opposing parts together, and constitutes *adhesion*. When this adhesion is completed, the new structure becomes a part of the living body.

Seeing, then, that the various changes are natural results of the operation of a cause, the practitioner must be careful to avoid doing anything that is calculated to irritate or debilitate the animal body. At the same time he must be careful to keep the excretory organs in a natural condition, maintain the strength by appropriate diet, and allay morbid irritability by the administration and application of suitable remedies.

In the first place, the practitioner is to avoid irritating and debilitating his patient. In order to accomplish this, three methods of treatment now pursued by many practitioners must be abandoned, namely, bleeding, purging, and counter-irritation.

*Bleeding and purging*, separately or conjointly, lower the animal powers, which are already too low, and prevent the physiological changes from taking place which are essential to the final removal of the disease. If they do not kill in a very short period of time, they cause an alteration in the inflammatory process, whereby large quantities of an aplastic material are formed, which, by blocking up the lung tissue, or filling the cavity of the chest, cause death by suffocation; and by their debilitating influence generally render the vital powers less able to resist even a mild attack of disease.

*Counter-irritation.*—This is the favourite method of treatment at the present time, and it is accomplished, or thought to be accomplished, by the application of mustard, cantharides, or other irritants to the skin of the sides and breast. It is applied on the principle that no two inflammations can exist in the body at the same time; that an artificial inflammation of the skin, excited by the irritant, removes or destroys that which is going on within the chest. Others say that it rouses the capillary circulation, removes congestion, &c.

For many years I followed this method of treatment, and so plausible were the arguments in its favour, that I could scarcely bring myself to believe that it could do harm, although I saw that many horses died even when a blister had acted. I was at last induced to abandon it altogether, and the result has been most satisfactory.

Irritation of the sides and breast is injurious in all instances of chest disease in the horse, except in a condition presently to be described, and for the following reasons:—

1st. It cannot and does not remove the internal inflammation. Many *post mortem* examinations have convinced me of this. During the summer of 1873 I had a good opportunity of demonstrating this fact to my students. A horse newly bought by a dealer died from pleurisy, and its value was in dispute between buyer and seller. On viewing the carcass, both myself and students were struck with the violence of the inflammation of the skin upon the sides, induced by the repeated applications of irritants. Some of the students, having been pupils of gentlemen who advocated the “counter-irritation” theory, were under the firm belief that no internal inflammation could exist where the external was so very marked. The result, however, proved the fallacy of the hypothesis, for the pleura was intensely inflamed. Still further, the editor has induced an acute pleurisy in healthy horses by applying strong blisters to one side of the chest.

The internal inflammation being an effect, until it can be proved that an external irritant can remove the cause which has produced such effect, it is quite as reasonable to suppose that a blister on the palm will remove or prevent the effects of another on the back of the hand, as it is to think that a blister on the outside of the chest, no matter how soon

applied, can remove an irritant, or prevent it from affecting the internal structures.

2d. Another argument which tells against blisters is the fact that their advocates differ as to the stage at which they ought to be applied. One party says they cannot be applied too soon, in order to counteract the internal inflammation, "by drawing the morbid action and the blood to the surface"; whilst another says that external irritants do harm whilst there is any fever present, and that they should always be applied after the pulse has fallen and when convalescence has commenced, in order to stimulate the removal of the products of the inflammation. I, however, fail to see the necessity of doing this; for the removal of the exudates is a physiological process, best accomplished when uninterfered with, and counter-irritation at this period can only retard the recovery, or, by again setting up the febrile condition, bring about a fatal termination.

There is, however, a very rare exception to these objections to external stimulants during the second stage of the disease, occurring in that condition where the horse neither gets better nor worse for several days—"hangs fire," as it is commonly termed. In such instances a weak solution of mustard, by gently stimulating the skin, will often induce the beginning of convalescence, and act as a stimulant to the whole body. In no other condition are counter-irritants beneficial, and in this, care must be taken not to apply them too strong nor for too long a time. The sides should be quickly rubbed over with about two or three ounces of mustard mixed in a quart of warm water, and the parts covered over with paper or linen. If one application does no good, it is not advisable to apply another; but if the horse seems to improve for a time and then relapse, a second or even third application may be admissible.

3d. The application of blisters causes pain and increases the fever. If applied to the sides they impede the respiratory movements by the pain they cause, and thus add to the distress and suffering of the animal. The cantharidine absorbed into the system causes irritation of the urinary organs, sometimes congestion of the kidneys, interferes with their excretory functions, and by the pain and disturbance thus induced adds materially to the febrile condition, and often causes the animal's death.

Horses that recover from the disease naturally do so much more rapidly than those which have been blistered, and which are often a long time in overcoming the effects of the blister; with raw sides, they remain for weeks in their stables unfit for work. Sometimes, more especially if the blisters have been oft repeated, the animal becomes much emaciated, is unthrifty, the blistered skin will slough, and when it finally recovers the blemish remains for life. Finally, animals which die from the disease do so much easier than those which have been severely blistered. Indeed, the agony of death from disease bears no comparison to that from treatment.

Having now pretty strongly condemned what I consider to be wrong in principle and disastrous in practice, I must proceed to describe the treatment which has proved successful.

During the premonitory fever, in addition to housing, clothing, &c., already described, the animal is to be allowed an abundant supply of cold water to drink, warm or cold bran mash, whichever it likes best, to eat, a boiled linseed mash every night, roots, such as carrots, turnips, or potatoes, with a handful or two of the best and sweetest of hay, or grass if in season. If the alternations of the temperature of the skin be very marked, two or three doses of spirits of nitrous ether are to be daily administered in warm water; and to excite the action of the kidneys, which is often in abeyance, the ether is to be supplemented by half-ounce doses of the nitrate of potash. Even when the fever is high and the symptoms acute my experience leads me to conclude that it is unwise to use depressants, such as aconite, antipyrine, &c. If the bowels are costive, clysters of warm water are to be administered, and in rare instances, where the bowels are extraordinarily torpid, a dose of linseed oil. In the majority of cases, however, laxative food will effect all that is necessary, and it is better to avoid even the oil, except where the constipation continues, or where it causes uneasiness.

The chest is to be carefully examined at least once a day, and when convenient the animal temperature registered, as well as the pulse and number of respirations.

If at the onset of the pleural inflammation there be pain and distress, manifested by the horse looking to its sides and grunting, or attempting to lie down, opium or morphia subcutaneously administered is to be given to alleviate the pain, and in order to

prevent its constipating effects, from eight to twelve ounces of oil are to be given. One or two doses will be sufficient, for when the dry condition of the first stage of the inflammation has passed away, pain generally disappears. If the symptoms are those of irritation rather than pain, bromide of potassium may be given in preference to the opium; and at any time during the progress of the disease, if irritability is much increased, the bromide will prove serviceable. In addition to the opium, warm, soothing—not hot, scalding—fomentations to the sides give great relief. These warm fomentations are to be applied for at least an hour two or three times a day in every severe case; and in all instances, whenever the breathing becomes catching or accelerated, they must be applied until relief is given. In many cases their effect in allaying pain and soothing the animal is most marked, the patient often giving a “sigh of relief” in a few minutes. The best method of applying them is either to wrap the horse’s body in a thick blanket or horse sheet, and pour warm water upon it, placing a tub so as to catch the water as it falls from the sheet, or to wring cloths out of warm water and apply them to the sides. The latter method is the cleaner, as there is less water lost on the bedding, &c.; but in the former the skin is not exposed during the fomentation, and is to be preferred. When the fomentation is concluded, the wet sheets are to be covered with a waterproof covering, or, if removed, the skin lightly rubbed with weak ammonia liniment, say one ounce of liquid ammonia to sixteen of oil, for the purpose of preventing the sensation of cold which is apt to follow warm applications.

The inhalation of oxygen, three or four times a day, has an extremely beneficial effect in many cases, and is in constant use in many practices, Mr. Faulkner of Manchester, the editor, and others, having great faith in its value.

As the local inflammation progresses the nitrate of potash is to be slightly increased. From one to two ounces may be given in the twenty-four hours, and it always acts best dissolved in the drinking water, as the horse can then sip it at its leisure. It lowers the animal heat, cleans the mouth, has some effect on the exudation, and stimulates the kidneys. When the diuresis becomes increased, the nitrate must be discontinued, and if the horse is progressing favourably, but

little more is needed. In the course of a few days, if the appetite is not good, vegetable, and afterwards mineral, tonics are to be given. In many cases where the kidneys act very languidly a few doses of colchicum will prove of great service. I think the colchicum seeds are the best, made into a tincture, and given in doses containing one to two drachms, for two or three days, or until diuresis is induced. The colchicum, in virtue of its action in causing the elimination of the solid ingredients of the urine, removes from the economy much effete material, products of the metamorphosis of the inflammatory exudates.

Some veterinarians are very fond of giving the carbonate of ammonia as a stimulant in all typhoid diseases. If the kidneys are acting freely it is a very useful stimulant; but if the secretion of urine is diminished, its administration only tends to load the system with ammonia—when it is already over-burdened with the products of tissue changes, all tending to be resolved to ammonia, carbonic acid, and water; but if administered in conjunction with the vegetable tonics in the later stages, it tends to stimulate the appetite and restore strength. It should always be given in a ball, as solutions tend to irritate the mouth, and prevent the horse from eating.

If the appetite is very much impaired, the horse must have an abundant allowance of milk to drink instead of water. Some horses are very fond of milk, and almost all will partake of it after tasting it a few times. If the debility is excessive, eggs beaten up with the milk will prove of great service. They supply those constituents which are required by the wasted tissues. If milk is refused, the animal must have gruel, linseed tea, or hay tea, if it will take them spontaneously, but nothing must be forced upon it.

Of the termination of pleuro-pneumonia in hydrothorax I have only to say that since I have abandoned the heroic and counter-irritating treatment, hydrothorax has been almost unknown to me. Other terminations, such as rheumatism, roaring, thick wind, &c., will be described under their proper heads.

## CHAPTER XL.

### ENZOOTIC AND EPIZOOTIC DISEASES—*continued*.

#### MALIGNANT CATARRHAL FEVER OF THE OX.

A MALIGNANT form of catarrhal fever, sometimes, but erroneously, called glanders of the ox tribe, due to the operation of a morbid poison, which expends its specific effects upon the mucous membrane lining the sinuses of the head and nasal chambers; manifested by rigors, dulness, and debility, succeeded by the mucous membranes becoming of a bluish-red colour, the eyes closed, the eyelids swollen, and flowing of tears over the cheeks. There is a painful and frequent cough; the pulse is feeble; bowels costive at first, but diarrhœa soon succeeds. In the course of a few hours after the onset of the disease, a profuse discharge issues from the nostrils, mouth, and eyes; the sinuses of the face and head become filled with purulent matter, and in some instances the *horns drop off*.

The connection between the bone of the horn (flints) and the sinuses of the head is very intimate, for the bony process, the horn core, which springs from the crest of the frontal bone is hollow, and this hollow is continuous with the frontal sinus. This hollow or sinus in the horn-process is very vascular, and its blood-vessels anastomose with those upon its periosteal surface. Any influence, then, which causes inflammatory action in the sinuses, easily induces the same processes in those structures which are so intimately connected; and such we find it to be the case in malignant catarrhal fever,—inflammation is excited in the sinuses of the flint, which extends to the surface of the bones, and the consequent suppuration detaches the horny coverings.

The symptoms of this affection are not dissimilar to those

of cattle plague. The discharge from the nostrils is more profuse than in the plague, purulent or sanguino-purulent, and *the horns fall off*.

There may be appearances on the mouth and in the visible mucous membranes very similar to those of rinderpest; and in some instances lameness and sloughing of the hoofs may lead one to suppose that it is an aggravated form of contagious eczema.

#### TREATMENT.

Removal to warm, sheltered sheds; enemas, to relieve constipation; stimulants, such as the carbonate or acetate of ammonia and the spirits of nitrous ether, with hot water vapour to the nostrils, constitute the treatment of the earlier stages. If the animal survive, these remedies may be succeeded by tonics, mineral acids, and good food. The disease is very fatal, and causes death in from three to seven days. If the animal live over the latter period convalescence may be expected. Violent remedies, such as strong doses of salts, bleeding, calomel, &c., always hasten the fatal termination. If aperients are imperatively called for, oil or treacle are preferable to the more drastic agents.



## CHAPTER XLI.

### ERUPTIVE OR PETECHIAL FEVERS,

USUALLY ASSOCIATED WITH EPIZOOTICS, ALTHOUGH THEY DO NOT THEMSELVES USUALLY ASSUME EPIZOOTIC CHARACTERS.

#### I.—PURPURA HÆMORRHAGICA, DIARRHÆMIA.

THIS disease was arranged in the first edition of this work in the same group as the charbonous affections, which in several characteristics it simulates; but the life history of the morbid processes, the causes, the non-contagiousness, and the absence of organisms in the blood in this disease separate it from anthrax.

Fault has been found with the term “*purpura hæmorrhagica*,” and Delafond has named the disease “*diastashæmia*.” I think the term is more objectionable than *purpura*, as it merely implies a standing apart or a separation of blood; and were I to venture upon a new term, I would suggest “*diarrhæmia*”—Gr. *δί᾽*, through; *ῥεω*, I flow; *αἷμα*, blood; or a disease characterised by breaking up of the blood, and ecchymosis.

*Definition*.—An eruptive non-contagious fever of an intermittent type, usually, but not uniformly, occurring as a sequel to another disease.

*Pathology and symptoms*.—The primary manifestations are often uncertain. In some instances the earliest symptom may be the expression of pain in one or more limbs, with but slight swellings, which, however, become more pronounced in a very short time. In other instances, the approach of the disease is shown by the appearance of a few petechial spots in the nostrils

or papulæ on the skin, whilst in other cases the malady is fully manifested in a very short period.

Similar to its analogue anthrax, purpura is a disease in which the blood is gravely altered, associated with extravasations, effusion of red coloured serum, preceded and accompanied by constitutional disturbance, languor, debility, sudden elevation of temperature to 104°-106° F., or even higher, the alternations of temperature being very great. The alternations in the following table are from the clinical report of a case which died at the College:—

Date.	Hour.	Pulse.	Temperature.	Date.	Hour.	Pulse.	Temperature.
1879, May 23.	9.20 A.M.	78	104.2°	1879, June 1.	6 P.M.	66	102.2°
„ „	6 P.M.	84	106.4°	„ 2.	9 A.M.	60	102.1°
„ 24.	9 A.M.	84	102.3°	„ „	6 P.M.	66	102°
„ „	6 P.M.	72	102.4°	„ 3.	9 A.M.	60	102°
„ 25.	9 A.M.	60	101.3°	„ „	6 P.M.	66	101.4°
„ „	6 P.M.	66	102.2°	„ 4.	9 A.M.	66	101.2°
„ 26.	9 A.M.	60	102.1°	„ „	6 P.M.	72	103.3°
„ „	6 P.M.	66	104.1°	„ 5.	9 A.M.	72	102°
„ 27.	9 A.M.	75	103.1°	„ „	6 P.M.	78	104°
„ „	6 P.M.	88	102.3°	„ 6.	9 A.M.	78	104°
„ 28.	9 A.M.	68	102°	„ „	6 P.M.	88	105°
„ „	6 P.M.	78	103.2°	„ 7.	9 A.M.	96	105.1°
„ 29.	9 A.M.	68	102.2°	„ „	6 P.M.	110	106°
„ „	6 P.M.	72	102.3°	„ 8.	9 A.M.	110	105.4°
„ 30.	9 A.M.	80	104°	„ „	10.30 A.M.	108	105.4°
„ „	6 P.M.	86	103.4°	„ „	1 P.M.	108	106°
„ 31.	9 A.M.	75	102.3°	„ „	3 P.M.	86	105.3°
„ „	6 P.M.	66	103.1°	„ „	4 P.M.	72	104.4°
June 1.	9 A.M.	60	102°	„ „	4.30 P.M.	Death.	

On the last day there was a copious discharge from nostrils and mouth. Horse kept on its legs till the last, and dropped down dead without a struggle.

The capillaries of the skin and mucous membranes are especially implicated in the congestions, and at all times there is a tendency to gangrene and decomposition of the extravasate, the products of which, being absorbed into the circulation, give rise to septicæmia and death. In some instances, thrombi form in the vessels of the parts more extensively and persistently swollen, and induce sudden death by embolism in the vessels of some vital organ, or a more protracted one by pyæmia.

In other instances, effusion poured out into the lungs is converted into a fibrinous exudate, which tends rather to a retrograde metamorphosis than to organisation, forming a caseous or semi-caseous mass, and interfering most materially not only with the integrity of the organ, but with the general health of the horse.

The more particular symptoms are petechial spots, of a dull mulberry or purple hue, on the visible mucous membranes. There is very often hæmorrhage from the mucous surfaces, the discharged blood being dark in colour and often fœtid in odour. Extravasations also occur in the substance of several viscera, more particularly the lungs, spleen, and liver. The limbs, lips, and other depending parts of the body swell, the swelling arising from extravasation of blood into the areolar tissue, and from the transudation of serum, and feebly coagulable lymph. The swellings are characteristic: they are generally uniform, extending perhaps the whole length of a limb, and terminating superiorly very abruptly, as if a cord had been drawn around the part. They are painful, hot, hard, and in those portions where the skin is barely covered with hair, as the lips, nostrils, and inside of the thighs, shining; and if uncoloured, petechial spots are seen upon its surface, but where it is dark-coloured no spots are discernible. Vesicles of about the size of a pea are present upon the lower parts of the limbs, around the hock and fetlock joints; these burst and discharge an amber-coloured acrid serosity that scalds and excoriates the surface of the skin over which it flows. Cracks and fissures appear at the flexures of the limbs, from which an unhealthy amber or purple coloured discharge issues. Swellings appear about the sheath, abdomen, and breast; these, in the earlier stages often disappear from one part of the body and reappear in another. In many instance the face, lips, nostrils, and eyelids become fearfully swollen; the swelling being tense, uniform, and ending abruptly about the forehead. This swelling of the head is apt to cause a fatal termination by interfering with the respiratory function; the swollen nostrils diminishing the calibre of the nares, great dyspnœa is thus induced. By interfering with the movements of the tongue and jaws it also prevents the animal from feeding, and thus becomes an additional cause of debility. The skin over the swollen parts has a great tendency to slough, leaving large open and unhealthy looking sores, which

discharge an unhealthy, foetid sanies. The pulse is feeble, often fluttering, dicrotonous—double, and there is great prostration of the vital powers. The bowels are at first generally constipated, but purgation is easily excited. Sometimes the fæces are mixed with blood, and abdominal pains are often present. In some instances there is soreness of the throat; but in purpura it is an accidental complication, and absent in the majority of cases. There is very often a hoarse or hollow cough, and a discharge of coffee-coloured fluid from the air passages. The petechial spots on the nose frequently slough, and leave a raw surface, from which issues an abundance of dark-coloured sanies, causing a snuffling sound in the nasal passages; and in some instances the tongue has been found swollen, with large, dark-coloured vesicles on its surface, containing much foetid material.

Some cases will commence to improve under proper treatment on the third or fourth day. Others take a much longer period, the fever being of a remittent character, and at all times the animal is liable to exacerbations from very trivial causes.

It is with difficulty that the animal can be made to move, owing to the stiff and painful condition of the limbs; it will often stand rooted to one spot for hours or even days together.

The urine is generally high, often dark-coloured, and emits a strong odour of ammonia; it contains much solid matter, and if kept in a vessel very soon undergoes decomposition.

In many instances the enteric complications are very grave and important, and in every case there is a danger of sudden extravasation into the intestinal mucous membrane and alimentary canal, the extravasations being so excessive as to cause rapid death from internal hæmorrhage.

*Causes.*—In the great majority of instances purpura occurs as a sequel to some debilitating disease, more particularly catarrhal fever, and its origin can in most instances be traced to bad ventilation or drainage. When an animal suffering from catarrhal fever is kept in a well-ventilated, well-drained stable, and otherwise properly cared for, purpura is scarcely ever seen; but if such an animal is housed in a badly drained, ill-lighted, and defectively ventilated stable, in fact, when it is compelled to inhale the products of decomposing urine, fæces, and of its own breath for several days together, the blood becomes so empoisoned with effete products that it loses its integrity, accumulates in the

capillaries and smaller veins of loose structures, more particularly in the depending parts of the body, constituting those swellings which are so characteristic of purpura, and when withdrawn from the body it is only feebly coagulable. Again, I have witnessed the occurrence of the disease in horses apparently recovered from influenza and strangles, placed to work or taken to exercise while still debilitated.

In rarer instances purpura may appear as a primary disease, traceable to defective drainage, ventilation, or bad food.

I have witnessed a few instances where death has occurred in the horse without the usual external manifestations; but where the *post mortem* examination revealed many of the characteristics of this blood disease, sometimes the animals have suffered from enteritis, sometimes they have sunk without manifesting any pain, have refused food, hung down their heads, the surface of the body being cold, they have become pulseless, and died in a few hours.

In one instance the animal suffered from several convulsive fits for three days, and died in one of them, the *post mortem* revealing darkness and fluidity of the blood, petechial spots on several internal organs, more particularly on the cerebro-spinal meninges. In this, as well as in the other cases, a careful examination of the tongue prior to death enabled me to detect the nature of the disease. This organ had a peculiar mulberry, purple, or claret colour, and that was the only symptom of blood alteration that could be detected, the Schneiderian membrane and conjunctivæ being merely injected.

It may safely be concluded that the causes of purpura are of a septic nature, and are due to the absorption of products of decomposition extrinsic to the body; to the severity and rapidity of tissue change within the body, either owing to a previous disease or debilitating circumstances, and to their accumulation when naturally generated, owing to impairment of the excretory organs.

The acute symptoms are generally of a remittent type, and when the practitioner is consoling himself with the belief that the animal is improving, it often happens that at the next visit the symptoms have become much aggravated, and the patient is rapidly carried off, dying perhaps on the second, third, or fourth day. In many instances again the acute

symptoms subside, and the animal begins to feed, but rapidly becomes emaciated, with great muscular debility, and inability to rise after lying down; these symptoms being accompanied by, or independent of, extensive sloughings of the swollen parts. When sloughing takes place, the skin and subcutaneous tissues assume a dry, leathery appearance, and separate tardily from the subjacent living structures; the discharge from the nose may continue, and the animal may die from repeated exacerbations, exhaustion, pyæmia, glanders or farcy, or from gangrene of the lungs, on the third, fourth, or even sixth week after attack. In some cases abscesses may be detected in various parts of the body, in the glands; or the lungs, liver, kidneys, &c., may be infiltrated with purulent accumulations.

In purpura, then, there is no doubt that the chief tissue of the body affected is the blood, the poison being a toxin which is absorbed. The inner lining of the blood-vessels is composed of a layer of cells whose vitality is derived from the blood passing through them, and any irritation of the blood will at once be felt by this layer of endothelium; the capillaries, being composed of this layer only, will necessarily be the quickest to suffer disintegration, and so we find that effusion of a serous fluid and ecchymoses are amongst the first clinical symptoms of the disease.

The *post mortem* appearances of those which have died during the early and acute stages of the disease are as follows:—

Darkness and fluidity of the blood; the subcutaneous tissues of the swollen parts filled with dark red or cinnamon coloured, feebly coagulable exudate. Dark spots will be found on the thecæ of muscles, and in the muscular structures of various parts of the body. They do not penetrate deeply into the muscular tissues, but the stain gradually decreases as the muscle is cut into, and some trace of it may be found to extend perhaps an inch into the thicker muscles. The various serous membranes—the pleura, endocardium, pericardium, and cerebral meninges—will be covered with petechial spots, the mucous membranes stained black or blackish-green. This colour will not be uniform, but interspersed with petechial spots, and in some instances the membranes will be covered with a thick layer of imperfectly coagulated blood;<sup>1</sup> and, again, cinna-

<sup>1</sup> I have a beautiful drawing, given me by Mr. C. Stephenson, Newcastle, of a brain and spinal cord of a horse which died comatose from purpura, showing the arachnoidean cavity, cerebral and spinal, filled with extravasation.

mon-coloured, gelatinous coagula will be found on the heart, along with a serous effusion, filling the pericardial sac. The dark spots become black on exposure to the air. The blood has a violet tint, and is variously altered on exposure; in some instances it reddens, in others, like the spots, it becomes darker. Kept in a vial closely corked, it will retain its fluidity for a very long period. (Healthy blood, to which ammonia has been added, can scarcely be distinguished from the blood of purpura.)

The mesenteric and other glands are generally congested, friable, and enlarged; the liver, spleen, and kidneys are variously altered,—sometimes merely congested, sometimes enlarged, softened, and containing a large quantity of dark-coloured fluid blood; the lungs dark, enlarged, pigmented, and more or less decomposed; the bronchi and trachea filled with dark-coloured extravasation.

*Treatment.*—It is useless attempting to treat this disease without first removing the animal from all offensive smells, bad drains, and bad ventilation. Pure air, light, warmth, and comfort are the first essentials. Secondly, it must be borne in mind that the great danger of a suddenly fatal termination is from extravasation into some internal organ, or even into the subcutaneous tissues. Some cases may die from the empoisoned condition of the blood, without the occurrence of any extravasation or even congestion, but these are rare compared with the first named.

For the purpose of overcoming this tendency to extravasation, styptics, such as turpentine or ergot of rye, or astringents, as the tincture of the perchloride of iron, are sometimes successfully employed. If these are given in combination with an oleaginous purgative, any astringent effect which they might otherwise exercise on the alimentary canal is prevented. It must, however, be remembered that extravasations depend more upon the condition of the blood than upon the blood-vessels, and remedies which are calculated, either by their antiseptic or oxidizing properties, to alter the abnormally fluid condition of the blood, are better calculated to attain the object than those which merely act upon an effect of the disease. For this reason, the chlorate of potash has been prescribed, and with marked results. Whether this salt acts as a direct antidote to the septic poison or not, is a matter which I cannot explain. It is a fact that blood, when drawn

from an animal suffering from purpura, or from one in a state of health, has its coagulating properties much increased when a small quantity of this salt is added to it. I have kept blood thus treated in a firmly coagulated condition for more than a year. The coagulation is so rapid and so firm that scarcely any serum is pressed out; and, what is most remarkable, there is but little contraction of the clot.

The action of chlorate of potash is to increase the coagulability of the blood, and so to a great extent limit the tendency to exudation. Its antiseptic property may also be efficacious, but that is of very little account as compared with its first action, which, by so assisting the blood, gives the animal organism time to make an attempt at elimination before it is too late. Calcium lactate might with advantage be tried, it being said to be the most useful drug.

Acting upon my experience as to the effect of the chlorate in promoting coagulation, and at a time when physiologists believed that fibrin was a natural constituent of the blood, I commenced to treat purpura with the chlorate, for the purpose, as I then supposed, of increasing the fibrin in the blood, and thus prevent the occurrence of congestions and extravasations. The result was most satisfactory; the swellings rapidly diminished, and restoration to health ensued in the great majority of instances. One ounce in the twenty-four hours is quite sufficient after the first dose, which may be from one-half ounce to an ounce, given at once. If more than this be given, intestinal irritation is apt to be produced, and the disease thus complicated. In addition to the administration of the chlorate, it is necessary that the excretory organs perform their functions, in order that the morbid material and the products of tissue changes be eliminated from the system; for this purpose oleaginous aperients are to be administered. The latest method of treating purpura is by the intratracheal injection of a solution of iodine, and to the success of which I can bear testimony from actual experience; in fact, the effect has been most remarkable, improvement taking place very quickly in cases which threatened to terminate fatally.

I do not recommend those strong doses used by some practitioners. The solution I use is made as follows:—Iodine 4 grains, iodide of potash 8 grains, water 1 ounce. I have not found it necessary to use a larger quantity than this in the twenty-four



hours ; the whole may be injected at once, or it may be divided into two injections daily.

Beyond this, I think medical interference is uncalled for ; and it may be stated here that much advantage is derived from non-interference, both in this and many other ailments of the lower animals.

The convalescence is very often prolonged, great prostration remaining for a long time. The animal must therefore have a liberal supply of good food—oats, beans, hay, &c.—and be carefully groomed and tended. The mineral tonics may be administered, and daily and carefully regulated exercise given.

*External treatment.*—If the head be much swollen, and there be a difficulty in breathing from tumefaction of the nostrils, continuous cold sponging must be ordered. The sponge may be dipped in cold water or in some weak astringent solution—vinegar and water, or the terchloride of iron tincture, largely diluted ; if the cold sponging fails to reduce the swelling, warm may be substituted, particularly if the weather be very cold ; but beyond doing this to the head, I am of opinion that all other interference does much mischief.

The swellings are but the result of the condition of the blood, and when the latter is restored to its normal standard the swellings will disappear. Indeed, an abatement of the swelling in one part of the body is often succeeded by tumefaction in some other, and perhaps more important structure or organ. The swellings are generally metastatic, and when they are confined to those parts of the body where they cannot endanger life, it is far better to leave them alone.

Puncturing the swellings is usually recommended and generally practised. In very severe cases of purpura admission of air into the degraded tissues causes deep and sometimes intractable sloughings. Punctures are, therefore, inadmissible.

If there is dyspncea from swelling of the throat or nostrils, tracheotomy must be performed, and it ought always to be done early, as it is essential that the horse be enabled to breathe freely ; in fact, it must not be forgotten that oxygen in abundance is required by the vitiated blood. The incision into the trachea should be antiseptically treated ; as there is a great tendency to gangrene of the exposed structures ; entrance of the gangrenous products into the bronchi, inducing septic pneumonia, thrombi in blood-vessels, pulmonary gangrene, and death.

## II.—SCARLATINA—SCARLET FEVER.

A febrile disease, characterised by an eruption on the skin, petechial spots on the nose or under surface of the lips, soreness of the throat, and sometimes suppuration in various parts of the body, particularly in the submaxillary space.

Unlike the scarlatina which attacks the human being, it is a non-contagious disease, generally attacking but one or two horses in a large stud, amongst which some form of epizootic disease is at the time prevalent.

Dr. Copland, in his *Medical Dictionary*, article "Skin," says—"First, That scarlatina was originally a disease of the horse, and that it formerly occurred, and has recently occurred, epidemically or as an epizootic among horses. Secondly, That it was communicated in comparatively modern times from horses to man. Thirdly, That it may be, and has been, communicated also to the dog." If this be true, scarlatina of the present time must be very different from what it has been in the past. I have seen numbers of horses suffering from it, but in no case did it exhibit any tendency to spread by contagion or infection; and I am of opinion that it is impossible to transmit it from the horse to any other animal.

Scarlatina is divided by medical writers into—(1.) Scarlatina simplex; (2.) Scarlatina anginosa; (3.) Scarlatina maligna; and (4.) Scarlatina latens.

Of these the two first are observed in the horse, what is called the malignant form being identical with purpura; it is quite possible, however, for any of the forms to degenerate into purpura.

Scarlatina is usually associated with epizootic catarrh, and occurs in animals that have been for some days suffering from that disease; and the production of such an alteration in the blood as induces the scarlatina is due to defective ventilation or drainage of the stable in which the animal has been kept, or to over-crowding, by which the air becomes loaded with decomposing animal matters. Sometimes a weak constitution will convert a catarrh into scarlatina, and the severity of an epizootic disease may alter the blood, and give origin to scarlatina.

Like chilblains, nettle-rash, and other affections where local swellings are the chief symptom, it is probable that this so-called scarlet fever is due to an excessive loss of coagulability of the blood. This is a common complication in chronic infectious diseases of animals, and people subjects of debility. In scarlatina the degree may be worse and more alarming, but the pathology would seem to be somewhat similar, in a minor degree, to purpura.

It has been undeniably demonstrated that there is absolutely no connection between human scarlet fever or scarlatina, and this affection in the domestic animals.

*Semiology.*—1. *Scarlatina simplex.*—On the third, fourth, or even as late as the sixth day after the commencement of epizootic catarrh, the animal is seen to be covered with blotches, upon the face, neck, body, and extremities. The blotches elevate the hair, but in many places there is scarcely any elevation of the skin, for if the hand be passed lightly over the apparent swellings, the skin is felt but little altered. In other parts of the body, particularly upon the inner aspect of the thighs, actual elevations of the skin in the form of rounded pimples can be both seen and felt. The nasal membrane will be covered with scarlet spots of variable size, and there will be a discharge from the nostril of at first a thin serous mucus, which afterwards becomes yellow or yellowish brown. The limbs are generally swollen, and the animal stiff in consequence. In some instances, no eruption is present, and the only evidence of scarlatina is found in the Schneiderian membrane, and perhaps the membrane of one nostril only will be covered with minute scarlet spots that escape the observation of all but the practitioner.

Soreness of the throat is almost a constant symptom, and if it has preceded the rash, it will in all probability be more or less increased; but it by no means follows that soreness of the throat will appear concomitant with the rash. In a few days the rash and scarlet spots begin to disappear, and on the rash declining, some desquamation of the cuticle takes place, the skin being scurfy, and the coat dirty for some time afterwards.

2. *Scarlatina anginosa.*—The symptoms at first may be those of the simple form, which, instead of disappearing, may continue to increase in severity. The limbs rapidly swell, the swellings

appearing in lumps or masses, large, hot, painful, and numerous. These swellings may also appear on the body and face; they are inconstant in their seat, often disappearing from one place and appearing at another, whilst the intervening spaces may be covered by a rash similar to that of urticaria, and by blotches not elevated to the touch. The skin covering the blotches will often be found moist, and an amber-coloured serosity will ooze from it. The petechial spots on the membrane of the nose increase in size, and become more intense in colour, assuming a tinge of purple, more especially at their centres; in other cases they coalesce, forming one large block, which covers the whole nasal chamber.

The soreness of the throat now becomes very great, and is accompanied by a loud moist cough, and at each cough large quantities of a yellowish-red mucus are discharged through the nose and mouth. There is difficulty in swallowing, and in some cases in breathing, the inspiratory act being accompanied by a roaring noise, and by a snuffling nasal sound. The submaxillary lymphatic glands become enlarged, tender, and inflamed, and suppuration occurs after the other symptoms have begun to disappear.

In favourable cases the soreness of the throat will recede with the eruption about the fourth or fifth day, whilst in others it will remain for some days after the disappearance of the rash. In all cases that recover, however, both the exanthem and angina will have disappeared by the ninth or tenth day, leaving the animal weak, emaciated, with swollen limbs, and in a more or less unthrifty condition for some time longer.

The fever is usually proportioned to the severity of the sore throat, and very often, but not always, to the rash. In slight attacks, the fever, rash, and angina are of a mild character; but in the severer forms they are proportionately grave. The pulse, which is always of a weak or feeble character, varies from 60 or 70 in the mild, to 90 or even 100 in the severe forms; the respiratory movements are accelerated, and very rapid when congestion of the lungs is present; the urine is scanty, and of a thick yellow or brownish colour, smelling strongly of hippuric acid, and quickly becoming ammoniacal; the bowels are constipated, but diarrhœa is easily induced.

Mr. Haycock describes the malignant form. I am of opinion

however, that what he thus describes is purpura, or a combination of purpura and scarlatina.

For a number of years I was of opinion that purpura and scarlatina were one and the same disease, but I have had occasion to alter this opinion, as typical cases of both diseases are not uncommon in Edinburgh. The distinguishing differences between the two diseases are to be found in the character of—*1st.* The petechiæ, which, in scarlatina, are composed of minute dots, forming a blotch by coalescence; the spots may be as large as in purpura, but each is composed of several smaller ones; they are scarlet in pure scarlatina, of a dark purple in purpura. *2d.* The sore throat. This is never absent in scarlatina, and but rarely present, unless it is a symptom of previous disease, in purpura. *3d.* The swellings. In scarlatina, these are at first in the form of lumps or masses, whereas in purpura they present an even surface, occupying the face, or a whole limb or limbs, and terminate abruptly above, as if a cord had been drawn tightly around the part. *4th.* Scarlatina is often associated with swelling and perhaps suppuration of the glands, whilst in purpura this is not present; and finally, the latter disease is characterised by a tendency to sloughing in various parts of the body, and occasionally by gangrene of some extreme parts of the organism, such as the ears, which will present a shrivelled and blanched appearance, become dry, and slough, leaving a raw, unhealthy surface. The animal temperature may not distinguish the one disease from the other. If scarlatina be severe, the thermometer may register 103°, or even higher; in purpura this would indicate a mild attack.

Scarlatina is sometimes followed by inflammation of the joints, which is supposed to be rheumatic in character, and by suppuration in various parts of the body; but I am not aware that it is succeeded by renal dropsy, or associated with albuminuria, as in the human being. There is generally more or less cedema of the limbs, breast, and abdomen, but these are not necessarily due to any renal complication, as they are common sequelæ to most debilitating diseases in the horse.

*Treatment.*—In the milder forms of the disease it is sufficient to keep the animal in a warm, well-ventilated, light, loose box, to feed it on laxative food, and give small and repeated doses of the nitrate of potash, hyposulphite of soda, or chlorate of potash,

in the food or water. The body must be clothed according to the weather, and the general comfort of the animal attended to.

In the graver forms, the animal must be carefully watched, more especially with regard to its breathing, for in some cases the glottis and other structures of the throat rapidly swell, the larynx becomes constricted, and the animal may die from suffocation. Whenever this is threatened, tracheotomy must be performed; and in all cases where the breathing is difficult, and accompanied by a loud roaring noise, this operation is to be resorted to, in order that the animal may obtain pure air to oxidize its already impure blood.

The throat is to be repeatedly bathed with hot water and enveloped in warm poultices, and the animal made to inhale the steam of hot water. The mouth is to be frequently washed out with salt and water, and if the coryza is excessive some of it may be applied to the nose. If the face and nares are swollen, they must be frequently bathed with cold water, and afterwards dressed with some astringent lotion, such as a solution of the tincture of perchloride of iron. If the bowels are very torpid, a gentle laxative may be administered, such as one pint of linseed oil, but active purgation must on no account be induced; the nitrate of potash may be given freely in the food or water, and abundance allowed of the latter, which must be cold and fresh. If signs of purpura supervene, the chlorate of potash must be substituted for the nitrate. If much depression is present, draughts of spirits of nitrous ether may be given, provided deglutition is not difficult; but if the act of swallowing causes pain and cough, nothing should be forced upon the animal, or suffocation may ensue. Indeed, where the throat is very sore, even the oil must be withheld, and the bowels relieved by enemas, or by the sulphate of magnesia dissolved in the horse's water. If he will drink this, it has a very good effect, but many horses will not drink at all if salts are dissolved in their water. After the first few days milk, or milk with eggs, is to be allowed in abundance, in order to support the animal strength. When the secretions are restored to their natural condition, and the soreness of the throat a little abated, a small ball containing carbonate of ammonia—the ball being well oiled—may be given two or three times a day with great benefit, and later on the mineral tonics, with bark or quinine. If soreness of the throat

and cough remain after the subsidence of the rash and fever, ulceration is to be suspected, and this can occasionally be detected by an examination with the oral speculum.—(See *Principles and Practice of Veterinary Surgery*, page 515.) It may be combated by a blister to the throat, or by direct application of the nitrate of silver solution to the part by means of a sponge tied to a piece of cane.

When suppuration has occurred, the abscess is to be opened, as the abscesses of scarlatina often become languid, and do not burst readily.<sup>1</sup>

Whenever the fever abates, and other signs of convalescence appear, food of the most nutritious quality must be allowed in moderate—not over-abundant—quantity.

Exercise should not be enforced until all febrile signs have disappeared, and the animal has to some extent regained its strength; even then much exercise is to be strictly forbidden. I have seen the most severe and rapidly fatal purpura caused by exercising the animal too soon and too severely.

I have said nothing about the treatment of the external swellings of the limbs. I am of opinion they should not be interfered with, as they are but expressions of a condition of the blood which does not endanger the life of the animal, and will disappear spontaneously.

The *post mortem* appearances are similar to those of purpura, with the addition of inflammation and swelling of the throat.

<sup>1</sup> The frequency of abscesses in scarlatina points to the conclusion that one or more of the cocci of suppuration is present—most probably the *Staphylococcus pyogenes albus*. As has already been pointed out, staphylococci are always present in the skin, and if any circumstances arise which will debilitate the animal, and so lower its resistance to bacterial invasion, there is every possibility of these staphylococci setting up local lesions upon, or in, the epithelium.

## CHAPTER XLII.

### SEPTIC BLOOD DISEASES.

DISEASES due to ordinarily non-contagious facultative parasites, but which become infective by inoculation in suitable soils.

The most deadly microbes are *Vibrio septicus* of Pasteur, the pyogenic bacteria, especially the streptococci, and some are sufficiently virulent to poison the most healthy; but generally the animal body whilst in a state of health is not affected by their presence, unless, indeed, they are very numerous.

Septic diseases are induced in two ways:—*first*, by the entrance of the microbes themselves into the general circulation (*septicæmia*), in which case the symptoms and results are not proportionate to the dose,—*i.e.*, fatal results may follow a very small dose; and *second*, by the entrance of microbial products—toxins only (*toxæmia*)—in which case the symptoms are proportionate to the dose,—*i.e.*, if large, the results may be immediately fatal; if small, it is soon excreted, and, having no reproductive powers, the effects soon pass off.

These microbes in their normal condition exist in external media, and only induce disease under circumstances which have rendered the animal body susceptible to the morbid effects of themselves or their ptomaines. For example, the body whilst in a state of health can generally resist the bacteria, unless very numerous; but when an organ or tissue injured—as in a wound—or diseased is exposed to the influence of such germs, they or their products overcome the resistance of the injured or diseased structures, penetrate the blood-stream, and give rise to grave disturbances, and even death. In proof of this I may refer to the experiments of Burdon Sanderson, who found that putrefactive fluids injected into the healthy peritoneal cavity of guinea-pigs produced no ill effects, but when the peritoneum had been irritated by an injection of some irritant, such as mustard and water, the virus penetrated the irritated membrane, inducing a general septicæmia, with rapidly fatal results.



Putrefaction is a form of fermentation, and as the yeast plant in alcoholic fermentation splits up the elements of the sugar into those of alcohol by a process of digestion, so the various putrefactive microbes consume the constituents of materials in which they are lodged, multiply rapidly when their food is abundant and suitable, alter its chemical combinations, the products of such change being excreted, with or without fœtor, in the form of various chemical combinations, S.H. and N.H. and other organic bases, designated *ptomaines*, some of which are injurious and some actually preservative. The writer's experience leads him to conclude that when the putrefactive materials are of vegetable origin, the products, subjected to sunlight and a plentiful supply of air, as in ponds, are generally innocuous, so far as the domesticated animals are concerned; but when they are of animal origin—cadaveric alkaloids, human ordure, decomposing animal fluids or solids, blood, milk, &c. contaminating drinking water, the results are often disastrous.

Many of my readers have repeatedly seen cattle standing in ponds during the hot summer weather for hours, polluting the water with their excreta, drinking freely of it, although in appearance it may resemble a green coloured gruel or porridge more than water. Yet they take no harm, indeed seem to thrive on it. If such water be examined microscopically it will be found to be swarming with bacteria of various kinds. But if such water contain the products of the decomposition of animal fluids or solids, even when sunlight and air are plentiful, there will be a repulsive odour, and unless forced, animals will not drink it; but when compelled for want of other supplies, the consequences are very prejudicial, the animals become emaciated, unthrifty, hidebound, often have diarrhœa, with irritation of the respiratory mucous membrane, causing the animal to cough, and if the water be not changed death may result. The *post mortem* will reveal a general anæmic condition, with bronchopneumonia, and perhaps some degree of congestion of the gastro-intestinal mucous membrane; as a rule, however, the lung lesions are in chronic cases more pronounced than those in the digestive tract. In some instances, and when the source of contamination is abundant, the animals may rapidly succumb to gastro-enteritis, with colliquative and fœtid diarrhœa, great prostration of strength, high fever—106-7° F. or more—and other

signs of putrid intoxication, arising from the absorption of the microbes or of the cadaveric ptomaine.

These observations only apply to the putrefaction of vegetable materials when occurring in ponds or ditches exposed to air and sunlight, for if the same materials are retained in leaky liquid manure tanks, and thus contaminate pump wells or other source of the supply of drinking waters, their effects are quite as disastrous as those emanating from animal sources. It is difficult to explain this fact, unless it be admitted that the algæ and other vegetations of low origin, which grow abundantly in ponds exposed to sunshine and air, either destroy the septic microbes or produce an anti-ptomaine, which overcomes the virulence by breaking down the toxic materials into simpler forms—ammonia, &c.

We cannot have a general septicæmia without the actual presence of the microbes in the circulation, and the pathogenic causative organisms are existent in the blood. As a rule, however, the blood does not seem to be a suitable medium for their development and growth; they soon disappear from it, are destroyed by the oxygen, or are arrested in the capillaries of the liver, spleen, kidneys, &c., and are there quickly destroyed; their virulence is thus overcome, and the disease is not transmitted from one animal to another.

In many cases, however, the germs remain external to the tissues, and act solely through their products, which gain entrance into the circulation and produce their virulent effects—toxic poisoning.

In many instances microbes or their products gain entrance into the body from without—traumatic septicæmia, septic metritis, &c.; but there are other instances where they are formed within the body itself, and microbes, always existent in the intestines, may give rise to systemic and local disturbances of a grave or even fatal nature.

The intestinal mucous membrane—always in contact with decomposing materials—has the power, whilst in health, of excluding them from entrance into the circulation; but when that membrane is irritated in any way and by any cause, it not infrequently loses that power of exclusion, and the ptomaines, indol, skatol, gaseous products, &c., instead of being eliminated, are absorbed into the blood, inducing high fever, local inflammation, diarrhœa or dysentery, and even death.

I think the above in a great measure explains why inflammation of the feet is such a frequent concomitant of bowel affections, even a slight irritation of the mucosa being sufficient to induce it, such as that arising from an ordinary purgative administered whilst the animal is in perfect health; absorption of gaseous products, as in flatulent colic; of putrefactive products in septic metritis; and in bronchopneumonia, when the catarrhal products have been retained in the bronchii, and there undergone putrefaction.

A form of poisoning arising from absorption of organisms or their products from the intestine has been named *stercoræmia* or *intestinal septicæmia*, and is said to supervene upon a number of conditions, such as insufficiency of gastric juice to neutralise the majority of the germs which pass through the stomach, &c.

In order to make this subject more easily comprehended, I have ventured upon the following arrangement:—(A) *Toxæmia*, where the products only are absorbed; and (B) *septicæmia*, where the microbes themselves, as well as their products, gain entrance into the circulation, and there increase and multiply. Under the first head may be included—

I. *Traumatic Fever*.—The mildest form of *septicæmia*. *Causes*.—Tissue necrosis, as in subcutaneous wounds, or in any effusion of blood, fibrin, or serum which undergoes but slight change from the mildly pathogenic staphylococci that are always present in the skin, and easily gain access through crevices in that tissue. They, in fact, find a favourable home in the skin and its appendages, but develop no lesions unless an injury takes place. Then normally the leucocytes are very quickly able to cope with them, and the inflammation they engender soon subsides.

*Symptoms*.—Sudden elevation of temperature—two to four degrees—as soon as shock has passed off; no other constitutional disturbance, the temperature falling in a few days, according to the extent of the absorption. If disturbance occurs, it is certain that some complication has arisen.

II. *Malignant œdema* (Koch), *gangrenous septicæmia* (Pasteur), is caused by the small sporogenous *œdema bacilli* called *Vibrio septicus* by Pasteur, which exist in large numbers, particularly in the upper layers of soil, and infection is easily

produced by subcutaneous connective tissue inoculation, but not by intravenous injection, or when applied to superficial scratches on the skin; the microbes, being anaërobic, are killed by the oxygen, and granulating wounds form an insuperable barrier. Chauveau says that a first infection confers immunity.

Some observers state that whilst the guinea-pig, goat, sheep, dog, horse, fowls, pigeons are susceptible to malignant œdema inoculation, cattle are absolutely refractory; but Kitt states that he has induced the disease in calves as well as in the other animals specified above; and further, that these bacilli are probably the cause of several diseases in cattle, such as septic parturition fever, surgical tumefactions, progressive inflammation of connective tissue, &c.

In the œdema of the septic centre the bacillus is found in the form of a rod with a spore at one end, or devoid of the spore, in which case it is of a slightly greater length; in serous membranes it reaches a considerable length, and becomes segmented into non-sporulated sections, and only invades the circulating blood towards the end of the disease or after death. They are little affected by antiseptics, but are killed by boiling for fifteen minutes and by a dry temperature of about 250° F., and Cornevin states that by continuing the action of heat and antiseptics he has obtained a vaccine which confers a safe immunity on animals, and particularly the dog, against septicæmia by them.

The bacillus is a rod measuring 4  $\mu$  in length by 1  $\mu$  in breadth, with or without a terminal spore, but, as already stated, it may be much longer in serosities; it is motile, having very active flexuous movements, quickly arrested by contact with oxygen, and slowly loses its virulence through the influences of putrefaction (two months, thus differing from the bacilli of Chauveau, upon which putrefaction has no effect). Chauveau and Arloing state that corrosive sublimate 1 to 500 does not kill it, and a three per cent. carbolic solution has no effect unless assisted by heat. Sulphurous acid, however, seems to have a rather powerful effect upon it. It is preserved when dried even at 100° F.

*Symptoms.*—Experimental inoculations in the connective tissues, particularly when protected from air, are followed by

an cedematous, doughy, sensitive, and crepitant swelling, extending in all directions, and there is intense reaction. The connective, adipose, and muscular tissues in the neighbourhood become the seat of gelatinous infiltration containing a large quantity of very foetid gases, and a reddish-yellow liquid teeming with the characteristic vibriones, absent in the blood during life, and only found in small numbers after death. The central part of the swelling soon loses its sensibility, and becomes moist, clammy, and cold—gangrenous; the circumferences of the swelling, however, are hot, tense, and extremely sensitive, the vibriones having evidently abandoned the centre, as only putrefactive germs can now be found therein, and invaded the surrounding tissues, where they continue to live at the expense of its vitality, and so on in a regular invasion until a general infection results, manifested by pulmonary oedema and congestion of the intestinal mucous membranes, the bacilli being found abundantly in the serous discharges, and but sparsely in the blood. The disease may follow surgical operations, particularly castration, when performed with unclean instruments.

III. *Stercoræmia*—*Intestinal Toxæmia*.—Absorption of bacterial products from any tissue or a mucous membrane, whether abraded or not—(a.) when the products are those of pyogenic bacteria, or (b.) those of ordinary putrefaction, e.g., *Proteus B. coli communis*.

*Symptoms*.—These vary in proportion to the dose. (a.) The temperature rises several degrees, and remains so till absorption ceases, from the products becoming arrested in the course of natural drainage, such as free suppuration, free incisions, and irrigation, when arising from a wound. Constitutional disturbance well-marked. (b.) When the dose is large, rapid collapse may follow.

(c.) *Surgical shock* is usually a septicæmia, in which death occurs in one to two days after severe symptoms, especially those seen after abdominal operations, castration, &c., owing doubtless to the enormous rapidity with which the peritoneum absorbs the bacteria and their products.

IV. *Hætic*.—A chronic form of sapræmia seen typically in long-continued “mixed infections,” in chronic tuberculosis, where there is a constant absorption of small quantities of virus.

*Symptoms.*—Irregular, with a tendency to periodicity of elevations and remissions, progressive emaciation, loss of appetite, and death from exhaustion.

#### PARTURIENT FEVER.

There are two distinct causes of *post-parturient sapræmia* and *septicæmia*—

1. *Retention of the foetal membranes, or "first cleansing."*
2. *Retention of the lochial discharge, or "second cleansing."*

In health, after a female is delivered of young, there is a continuance of labour pains, known as after-pains, and as a result of these efforts the uterus contracts, and at the same time any portion of foetal membranes which did not accompany the fœtus are expelled.

In most of the domesticated animals retention of these membranes for many hours results in grave lesions. The membranes, portions of which may protrude from the vulva, become infected with various organisms. These grow rapidly, and penetrate to the portions of membranes in the womb. The amount of toxin excreted by them is enormous, and if it be absorbed it gives rise to septic intoxication. In many cases the organisms themselves gain entrance to the wall of the uterus, and then there is a septic metritis, followed by a septic metro-peritonitis.

The mare seems far more susceptible to this affection than any other of the domesticated animals; the cow less so than the rest. Retention of the membranes in the mare for a few hours may result not only in a septic metritis, but also in a septic laminitis and pneumonia, whereas in the cow retention may have been for days, and yet the animal feeds and milks, and has none but local symptoms.

It is obvious from the above remarks that after a mare has foaled the membranes should be at once removed, and that in the cow one may with comparative safety leave matters to nature for a day or two.

When an animal becomes affected, the first symptom is usually a more or less difficulty in standing, a swinging of the hind quarters, and a tendency to fall; the appetite fails,

the secretion of milk is suspended, respirations are hurried, pulse quickened, and temperature elevated. There are often strainings and expulsion of small quantities of dark coloured discharge of a most foetid odour. On further examination, the lips of the vulva may be puckered, the vagina hot and red, and considerable pain evinced if the os uteri be touched. If the surgeon passes his hand and arm right into the womb, he finds it distended, and containing considerable quantities of fluid and portions of foetal membranes.

If an examination be made *per rectum*, the uterus can be felt as distended and with hardened walls.

It is not until impending death that signs of delirium appear; and though there may be a paralysis of the hind quarters, there is never that comatose condition and complete loss of volition as seen in parturient apoplexy.

With regard to treatment, if possible remove the patient to a clean loose box, and have a good bed of straw put down; then, after having smeared the hand and arm with carbolised oil or vaseline, remove all remaining portions of foetal membranes which have remained in the womb. This must be succeeded by tepid injections of antiseptic solution, to be repeated twice or three times daily.

As a matter of self-protection, it is advisable to thoroughly irrigate the womb prior to the removal of the membranes.

The patient must be kept warm, and if necessary backed, and the urine withdrawn by means of a catheter.

The medicinal treatment should be of an antiseptic character—hyposulphite of soda, quinine, turpentine and oil, and such-like medicines. Saline purgatives and absorbents are contra-indicated. The strength should be maintained with eggs, linseed, and other mashes, and easily-digested nourishing foods.

*Post mortem* appearances indicate a general septicæmia, with enlarged, hardened, and inflamed uterus, containing quantities of foetid material.

When the disease is due to the retention of the lochia, we have the history that the first cleansing, or foetal membranes, had been successfully cast or removed; further, that it is several days since parturition; and, finally, on examination through the vaginal canal, we find the os uteri closed, and

so retaining the lochia, which had become infected prior to the closure.

The cause of the two affections is the same—namely, decomposition and septic infection of the contents of the womb—and the line of treatment is the same in both. In the latter form it may be necessary to use considerable pressure with the fingers to open the os uteri.

#### BRAXY.

*Septicæmia Gangrenosa.*—*Braxy.*—*Striking of Blood, &c.*—This is a well-known disease of sheep, particularly of young one-year-old sheep—hogs—prevailing during the autumn and winter months, and is known in Scotland under two different terms—namely, “dry braxy” and “wet braxy,” the latter form being characterised by more or less effusion of a reddish-coloured serosity into the peritoneal cavity. This latter condition is to be looked upon as an endeavour to excrete the morbid material from the circulation, and may be compared to the compensatory pleurisy and hydrothorax witnessed in septic broncho-pneumonia subsequent to parturition, or as a result of the ingestion of food containing ptomaines of animal origin.

*The Symptoms.*—Unfortunately in the majority of cases a sheep left quite well at night is found dead in the morning, and the shepherd judges of the nature of the disease by the swollen and discoloured appearance of the carcase. But if seen some time prior to the fatal result the first symptom seems to be manifested by a short step, then the animal stands apart from its companions with the head depressed and back arched. Tympanites rapidly follows, the stomach becoming enormously swollen and distended with gases, and is resonant on percussion. The other signs are those of colicky pains, lying down and rising alternately, or standing apart with the head and ears hanging down, the eyes dull, and the back arched; prostration and swelling of the belly rapidly increase, the sheep then goes down, and, although struggling, is unable to rise, when death closes the scene.

The *post mortem* appearances are analogous to those seen in cattle, namely, a great tendency to rapid decomposition of the



blood and tissues; the blood-vessels are full of dark semi-fluid blood; bloody froth issues from the nose, mouth, and sometimes anus; the flesh has a dark red appearance; petechial spots are visible upon the serous membranes and in the subcutaneous tissues; the abomasum and duodenum are highly congested, and covered with large dark-coloured spots of ecchymosis; the alimentary matters are mixed with blood, and the mucous membrane generally infiltrated in its substance, and coated on its surface with a layer of semi-coagulated, dark-coloured blood. The peritoneal cavity is generally filled with serum, and the bowels distended with foetid gas.

The pathology of braxy has yet to be determined, but it seems to be one of the forms of *mycotic gastro-enteritis* arising from the ingestion of grasses in a semi-putrefactive condition, as it is only commonly seen after the grasses have been more or less destroyed by frost, and is most common in frosty rimy weather, or when a hoar frost has covered the ground. When grasses in this half-frozen and withered state are eaten, the stomach, debilitated by the coldness or low temperature of its contents, is to some extent paralysed, and consequently disabled from performing its function. As its contents become warm, fermentation rapidly takes place, ptomaines and noxious gases are freely evolved, which, gaining entrance into the circulation, set up a fatal septic or putrid intoxication. In order to prevent this fermentation, I would suggest that, where it is possible, the sheep should be fed on hay in the early mornings, in order to moderate the keenness of the appetite, and thus ensure a slower and more perfect mastication; and also, by elevating the temperature of the cold food, prevent lowering the temperature and vitality and function of the digestive organs. When sheep are folded on turnips, "sickness" might be prevented if this were done.

*Treatment.*—Unfortunately there is seldom any opportunity of testing the efficacy of any remedy, but should there be an opportunity, the hyposulphite of soda might be administered, say in half-ounce doses, dissolved in half a pint of water, for a well-grown hogg or sheep. This remedy counteracts putrefaction, and has a slightly aperient effect on the bowels. Carbolic acid or any of the other disinfectants are also serviceable, and to prevent death from suffocation the rumen should be punctured, and the gases allowed to escape.

The flesh of sheep which have died, or been slaughtered whilst on the point of death, is generally salted and dried—made into mutton ham—and eaten with impunity in many parts of Scotland, and it can be truly said that it is at least equal, if not superior, to venison, and any one who is fond of high game would thoroughly enjoy a nicely brandered cut of it.

Septicæmia and pyæmia in young animals are known under the terms pyæmic omphalitis, navel-ill, pyæmic arthritis, joint-ill, erysipelatous disease, also called black spaud in Scotland. Omphalitis and arthritis will be found described in my *Veterinary Surgery*.

Even a more marked form of malignant cedema or gangrenous septicæmia prevails and is known in Scotland by the term *black spaud*, a gangrenous form of disease seen in lambs under a week old, and characterised by the skin and subcutaneous tissues surrounding and extending from the umbilicus, becoming slightly swollen and assuming a dark purple colour. The discoloration and swelling are sometimes in large patches, covering the lower parts of the abdomen, breast, and sides, but more frequently they consist of one large diffuse patch, embracing the regions mentioned, and extending to one or both shoulders, hence the term black spaud (shoulder).

This disease sometimes commits great havoc, the author having known as many as ten score lambs being carried away at one sheep farm in a few days.

*Etiology and Pathology.*—If during the first few days of the lamb's life, or before the umbilicus has completely closed, and a hard eschar formed, the umbilical cord be brought into contact with decomposing materials, both the germs and the products of the septic process may gain entrance into the umbilical canal, and there, acting upon its contents, set up the putrefactive process, which, extending into the surrounding tissues, induces locally congestion and thrombosis of the blood-vessels, characterised by diffuse mulberry coloured patches, with or without suppuration of the cord, and generally great and increasing prostration, and a rapidly fatal collapse, death occurring in a few hours after attack.

On many farms the sheep slaughtered for food are skinned in the lambing sheds, the blood and offal allowed to remain on the ground, the skins thrown across the beams of the roof to dry; and the skins of others which have died of disease or accident

are also brought there, and allowed to remain there, very often until the next clipping season.

From the foregoing observations it is evident that infection of the umbilical cord by the products of putrid decompositions which cover the floor of lambing grounds, particularly lambing sheds, is the starting-point of the infection.

Can it be wondered that lambs dropped in such an atmosphere fail to escape the contamination?

Careful observers, whilst agreeing that permanent lambing sheds, contaminated as they necessarily must be by decomposing materials, are the great cause of both black spaud and joint-ill, state that both of these forms of septicæmia are more prevalent during cold east winds; and they reason as follows:—1st. That during cold weather the ewe does not cleanse so freely and quickly—indeed in some instances, owing to the debility induced by the cold, expulsion of the foetal membranes does not occur; that the retained membranes putrefy, giving rise to foetid discharges and emanations: 2d. That in order to receive warmth the lamb clings more closely to the mother, and is consequently more exposed to become infected by the septic matters. Many who have experienced losses from this disease have now dispensed with permanent lambing sheds, and lamb their ewes in an open but well sheltered spot, or in temporary removeable sheds.

*Treatment.*—The disease is not amenable to medical or surgical treatment, therefore its prevention should be carefully studied.

All lambing sheds should be abolished; but if this is impossible, they should be thoroughly cleaned, lime-washed, and disinfected before the commencement of the season, all skins and decomposing materials being first removed. The bedding should be dry, frequently removed and replaced; all blood, foetal membranes, &c. swept away at the end of each day, and before putrefaction has commenced, or else the place will soon become tainted. If the disease has made its appearance, the ewes should be lambed somewhere else. If in the absence of sheds the lambing take place in the open air, the ground should be clean and protected from cold, particularly east winds, as these are acknowledged to predispose to the disease, and some anti-septic dressing, such as carbolised oil, or sublimate solution, applied to the umbilicus.—(See *Joint-Ill, Veterinary Surgery.*)

Septic diseases may become contagious amongst animals under certain conditions, as in metritis of the ewe. Here the initial or spontaneous attack spreads amongst lambing ewes, but has no effect upon the males and barren females.

#### SEPTIC PNEUMONIA OF CALVES AND LAMBS.

This disease has prevailed to a considerable extent in various parts of England, Scotland, and Ireland during the last few years, committing great havoc amongst calves a few weeks or months old, killing them very quickly. Poels, who has studied this disease, states that it is due to the presence of a facultative bacterium which is found in the soil of certain farms, and the disease is thus rendered persistent on such infected lands. It is an ovoid bacterium with rounded ends, with a clear centre, measuring from  $\frac{1}{5000}$  to  $\frac{1}{15000}$  of an inch in length, and  $\frac{1}{5000}$  in breadth, staining with aniline colours, but not by Gram's or Weigert's. The same, or a similar bacterium, is found in the broncho-pneumonia of American cattle. The same microbe is said to be found in the diarrhœa of calves—"white scour"—and calves fed with small doses of bouillon cultures, about  $1\frac{1}{2}$  drachms, become diseased, and die in one to two days. The same germ is found in the contents of the intestines of healthy calves, but destitute of pathogenic properties, and it is supposed by Jensen that it only becomes virulent under certain abnormal conditions, perhaps attributable to the diet, and that this virulence becomes increased by subsequent passage from calf to calf.

The symptoms are those of pneumonia, with or without diarrhœa.

There is a cough, with discharge from the nostrils, and the catarrhal products contain the microbes, which are thus spread around with the expectoration and fæces. Anyhow, it is admitted that the disease is transmitted from one animal to another, and Poels says that he has found the microbes in all the organs; and he is supported by Jensen, who has found them in the blood, spleen, liver, lungs, kidneys, heart, and in the mucous membrane of the intestinal canal.

The disease is occasionally very fatal amongst sheep, particularly lambs and others under one year old (hogs), and

Galtier states that it is identical with pneumo-enteritis of the pig, and that he has transmitted it from the pig to the sheep, guinea-pig, rabbit, and from these latter he has retransmitted to the pig and sheep. He has also transmitted it to the dog, goat, calf, donkey, and poultry, and to a goat in an advanced state of pregnancy, inducing abortion. The foetus when examined was found to have in the thoracic and abdominal cavities a sero-sanguineous exudate, containing the microbes, and reproducing the disease by inoculation.

Since Nocard found that "white scour" in Ireland was caused by a *Pasteurella* (bipolar organism), much more attention has been devoted to these parasites. It is not now by all observers accepted, that white scour, joint-ill, and lamb-sickness, are invariably due to these organisms; in fact, from the recent researches of Jensen, it would seem that organisms of the colon type are largely responsible, and Baldrey found only *coli* in an Indian outbreak. Jensen has made a polyvalent vaccine from colon organisms which he has found to be of exceeding benefit in preventing this complaint, and this again would point to the colon bacillus being the main factor. On the other hand, Lignières in South America, and Bowhill in South Africa, think that the *Pasteurella* is the cause of joint-ill and *Lamzickte*, or lamb-sickness; and Lignières has made a polyvalent protective vaccine and a polyvalent curative serum from these bipolar organisms, which have also given good results in the Argentine. It would seem, then, that both organisms may play a part, and either may produce very similar symptoms. It has already been pointed out that a *Pasteurella* may induce an acute disease, but it may be impossible to detect the organism microscopically or culturally as in the case of contagious pneumonia. If a colon vaccine will induce immunity, then the disease in such an instance is certainly not a *Pasteurellosis*, and *vice versâ*. The question as to cause, then, remains in some doubt.

The *post mortem* appearances are those of intense pulmonary congestion, with or without pleurisy. In some cases a gelatinous exudate is found on the pleural surfaces, whilst in others they have simply presented a more or less deeply congested appearance. On cutting into the lung structure, minute yellowish-white spots are to be seen, and if the lung be pressed, a muco-

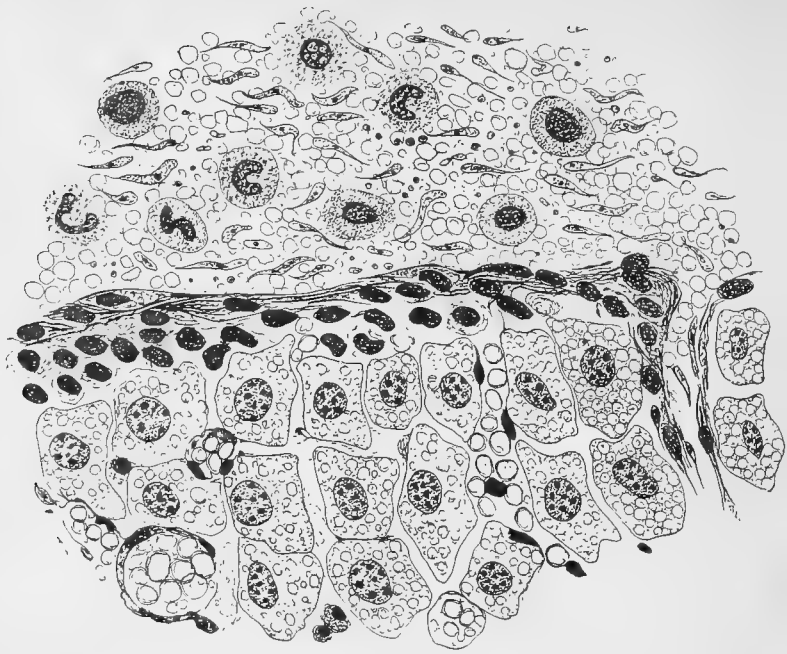
purulent discharge will be seen to issue from the inflamed bronchi, and if this discharge be examined microscopically it will be seen to contain the microbes more or less abundantly.

The pericardium, heart, liver, kidneys, and digestive tract will also often be found more or less inflamed.

*Treatment.*—Assuming that the usually harmless microbe becomes virulent under abnormal conditions of the body of its host, and in order to limit the spread of the disease, separation of the sick from the healthy should at once be resorted to, and the surroundings cleansed and disinfected. Medicinally but little can be done beyond endeavouring to destroy the virulence of the microbe by the administration of carbolic acid, hyposulphite of soda, or remedies having a similar effect, as the condition of the animal might justify.



PLATE XVIII.



1.—SECTION OF LIVER OF MOUSE INFECTED WITH TRYPANOSOMIASIS, SHOWING THE PARASITES IN A BLOOD-VESSEL.



2 -TRYPANOSOMA EQUINUM IN BLOOD BEFORE TREATMENT OF THE PATIENT WITH ATOXYL AND TRYPAN RED.

From 'Trypanosomes, Trypanosomiasis, and Sleeping Sickness: Pathology and Treatment,' by Wolferstan Thomas and Anton Breinl, in Memoir XVI., Liverpool School of Tropical Medicine.



3.—TRYPANOSOMA EQUINUM AFTER TREATMENT.



## CHAPTER XLIII.

### BLOOD DISEASES.

#### TRYPANOSOMIASIS.

TRYPANOSOMIASIS is the term applied to a numerous class of specific diseases, due to the presence of hæmatozoa of the genus trypanosomes in the blood of the affected animal.

*Trypanosomes* are morphologically recognised as being somewhat fish-shaped, each having a flagellum on the anterior end. They are endowed with life, and are of the animal and not of the vegetable world, and are classed as protozoa.

If a trypanosome be stained by Romanowsky's method, we find the body to be coloured blue; in the anterior end we see a red-stained nucleus, and at the posterior end a much smaller red-stained body, sometimes called the nucleolus, but better known as the centrosome.

From this centrosome a thin red thread extends along the outer margin of the body right forward to the anterior end, where it becomes continuous with the flagellum.

Trypanosomes multiply by longitudinal fission; first the centrosome, then the nucleus divides, and then a new flagellum appears; it often happens that these young trypanosomes remain slightly attached to the parent, and thus give rise to the roseate appearance.

Koch divides trypanosomes into two great groups, and this grouping is based on three important peculiarities—the morphology of the parasite, its virulence, and its relation to its host.

The first group only exists in one species of animal; their virulence is constant but slight, and the group comprises the trypanosome of rats and the *Trypanosoma Theileri*.

The second group, which includes all the other forms of

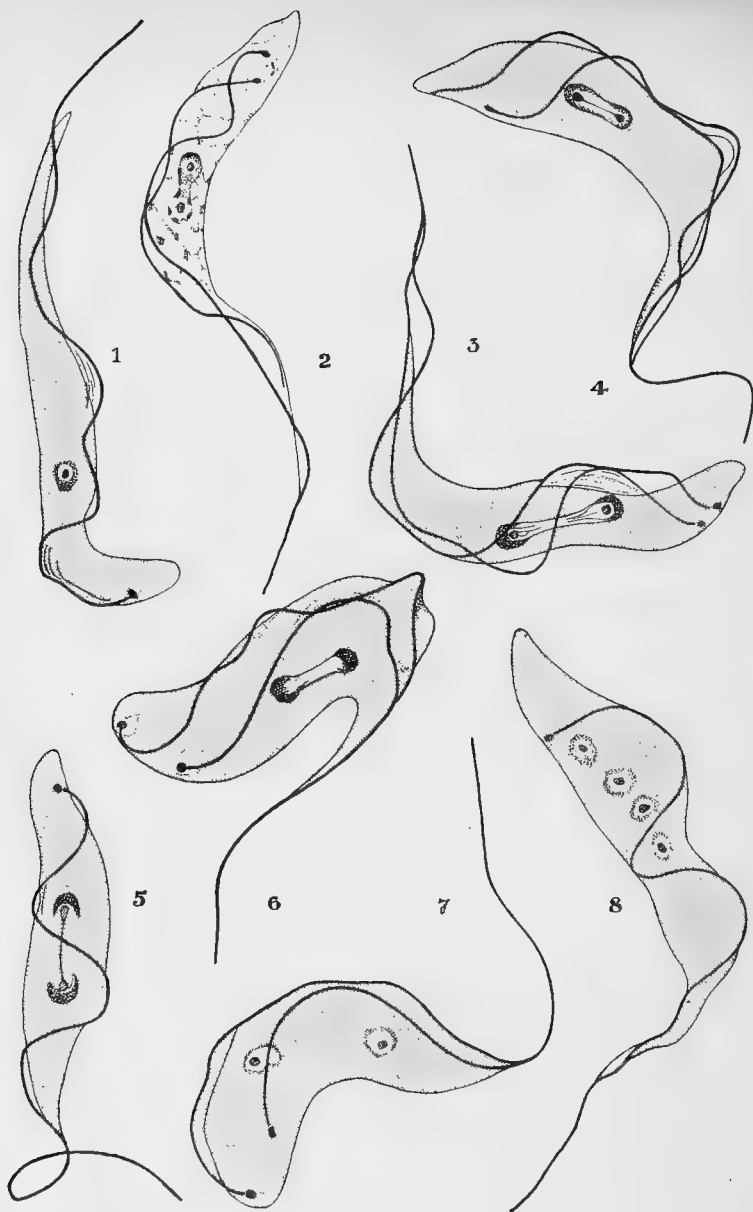


FIG. 38.

1, Resting stage of the trypanosome ; 2, stage showing formation of the new flagellum and division of the nucleus ; 3, trypanosome showing details of the division of the intranuclear centrosome and the nuclear substance ; 4 and 5, trypanosomes showing later stages of the same process ; 6 and 7, trypanosomes showing still later stages in the division of the nucleus and the characters of the intranuclear centrosomes ; 8, trypanosome wherein the nucleus has divided into four constituents, although there is only one flagellum.

trypanosomes, exhibits great variations both in form and in virulence, and is not confined to one species of animal, but may affect horses, dogs, rats, &c., and what is of considerable interest, individual species may morphologically differ in different animals. Not only do they vary morphologically in different animals, but particular trypanosomes vary in virulence in animals of the same species.

It is further shown that trypanosomes which have great virulence in some animals, have very little when inoculated into other animals, or gradually become modified in their virulence if passed through a series of animals of the same species.

Thus, by inoculating dogs with trypanosomes of comparatively small virulence, and reinoculating from dog to dog in series, we find that the virulence increases in each stage. Again, trypanosomes which are very virulent to oxen if introduced directly, are much reduced in virulence if first passed through rats and dogs.

Many animals harbour trypanosomes which are seemingly harmless, but which if passed into other animals will give rise to virulent disease.

The trypanosomes which are of most interest to veterinary surgeons are the causes of the following diseases, which will be discussed separately :

Mal de caderas, *Trypanosoma equinum* (has no centrosome ?) ; surra, *Trypanosoma Evansii* ; tsetse-fly disease (nagana), *Trypanosoma Brucei* ; dourine, *Trypanosoma equiperdum*.

It may be well to state that at the present time the leading authorities are agreed that *mal de caderas*, *surra* and *nagana* have many symptoms in common, but there are these differences :

1. Animals immunised from *nagana* may contract *mal de caderas*, but not *surra*.

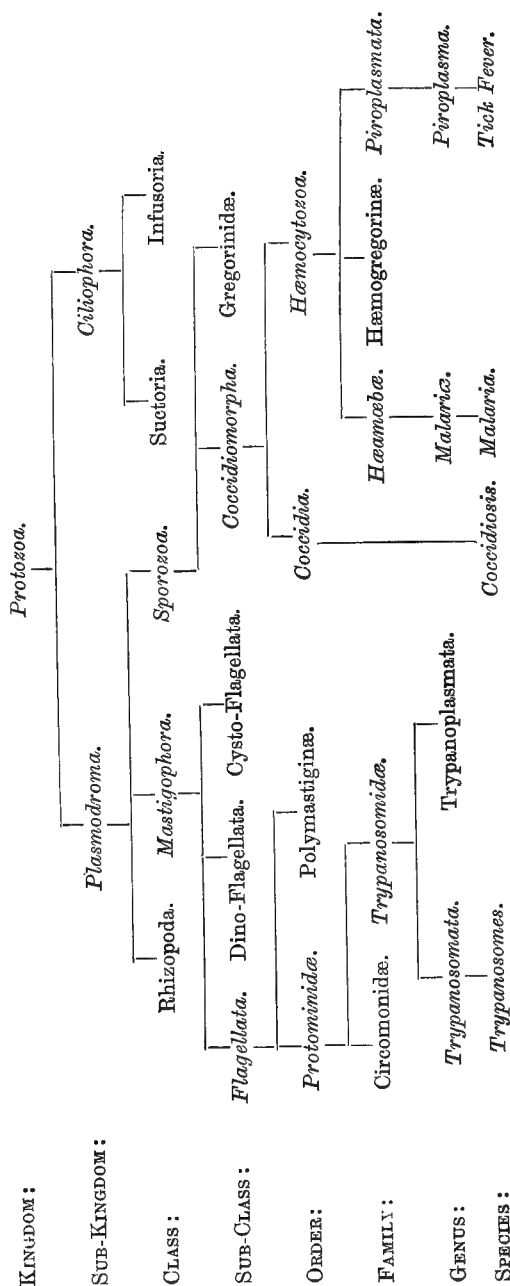
2. In *mal de caderas*, hæmoalbuminuria is almost a constant symptom, but it is an extremely uncommon one in *nagana*.

3. In *mal de caderas* paralysis is a most marked symptom, but it is rarely seen in *nagana*.

Appended is the genealogical table showing the origin of the trypanosomes.

## CHART.

## CLASSIFICATION OF PROTOZOA.



The families that are printed in italics are those having a pathogenic interest.

## MALADIE DU COÏT.

The name *maladie du coït* gives no further idea of the disease than that it results from the act of copulation. Other and various denominations, however, have been offered as expressing more definitely the character of this affection, some having been founded on its supposed analogy with the syphilis of the human being, and others upon the predominance of certain symptoms which have most particularly struck the observers.

Among these designations are the following:—*Syphilis du cheval* (syphilis of the horse); *vérole*; *typhus vénérien*; *maladie pustuleuse et chancreuse maligne* (frambœsia morbus pustulosus) (Erdley); *épizootic chancreuse* (Hering); *maladie vénérienne du cheval*; contagious eczema; paralytic disease of the horse; epizootic paralysis; epizootic paraplegia (Signol); dourine, &c.

## DOURINE (MALADIE DU COÏT—HORSE SYPHILIS).

Due to the *Trypanosoma equiperdum*. These parasites are found not only in the blood, but also in other fluids of the body, and also in the discharges from ulcers.

The *Trypanosoma equiperdum* is smaller than that of surra, being about 18 to 26  $\mu$  in length and 2 to 2½  $\mu$  in width (Baldrey).

*Infection* is conveyed from animal to animal of the equine species, usually during the act of coitus (inoculation from an infected sponge or infected litter is improbable, if not impossible, as the protozoan parasites would be too weakened or even killed by such treatment) and, it is said, not by any other natural means; thus differing from other known forms of trypanosomatic disease, which are conveyed from animal to animal by means of flies.

The disease may be induced in the ass, rabbit, rat, and mouse. The ox is said to be immune, but may have borne the parasite without showing any clinical symptoms.

The period of incubation is about seven to fifteen days after infection from coitus; it is less if artificially induced.

After the period of incubation, the first symptom noticed

is, in the male—urethral irritation and a discharge—and in the female a discharge from the vulva.

Nocard divides the progress of the disease into three stages:

*Primary*, in which there is urethral or vaginal irritation, with ulceration and discharge, and frequent attempts to urinate. The mucous membranes of the genital organs become reddened and swollen; and owing to this irritation the horse has frequent erections, and the mare shows signs of œstrum at short intervals.

As the disease progresses the stallion has phymosis and paraphymosis, and œdema of the dependent parts of the body—more particularly the testicles and scrotum.

Often the ulceration may extend to the sheath and the scrotum, but yields to ordinary treatment, only, unfortunately, to break out again. During this, the primary stage, there is little or no fever, and the appetite remains good; in fact, in many cases the appetite is voracious, though all the food taken is, as they say, “put into a bad skin,” and the animal pines.

*Secondary*, four to six weeks, in which there are marked skin lesions—notably, an exanthematous condition of the skin and the appearance of “plaques,” which look much as if odd shillings and half-crowns had been slipped under the skin. These patches come and go in a very indefinite manner. They exude a sticky substance, and sometimes become œdematous and then persist. This condition is always accompanied by progressive anæmia and by involvement of the various lymphatic glands—more particularly those of the groin, which may suppurate at this stage. Owing to debility and neurotic affection in its initial stage the animal rests a great deal, is most apathetic, and has difficulty in rising, and when on its feet its power of progression is uncertain, and wobbly if trotted.

Stallions and mares from the commencement of the disease are usually sterile, but if a mare becomes pregnant she is almost certain to abort.

*Tertiary stage*, characterised by grave changes in the nerve centres, and terminating in paraplegia or partial paralysis, often of one leg only.

In this stage of the disease the patient is very emaciated,

there is more or less fever, the appetite is capricious, the mucous membranes now assume a pale pink colour, the urine is irritant and thick, the power of co-ordination is greatly interfered with, and it is with the greatest difficulty that the animal manages to even support itself, far less progress. Indolent abscesses appear in the scrotum and sheath, and, if it be possible, the animal gets still more shadowy and dies.

During the whole course of this disease, which may extend over a period of eighteen months, or even more, it should be noted that it is an intermittent disease, having exacerbations as each new crop of parasites is born; but there is little elevation of temperature at this period, and there is not that marked subnormal temperature after the exacerbation as is seen in other trypanosomatic diseases.

The disease might be confounded with farcy, but the mallein test would determine that.

Again, the disease, being transmitted by coitus, is only seen in stallions and covered mares; it never occurs, naturally, in geldings or virgin fillies.

Pease finds that a ready means of differentiating between surra and dourine is to inoculate a pariah dog with some blood from the patient. If it be dourine, the dog remains healthy; if surra, the dog becomes ill from surra.

#### GENERAL SYMPTOMS.

As these are the same in both sexes, it would be useless to consider them separately.

A more or less abundant discharge of a muco-purulent matter from the nostrils, and of tears from the eyes, sufficiently indicate that there is a general affection of the mucous membranes, of which those visible are highly injected.

The lymphatic ganglia, not only in the vicinity, but also those in the submaxillary space, are engorged.

The appetite is generally good, and the food thoroughly digested; nevertheless, the animal gradually falls off in condition, and, to use a common expression, becomes hide-bound, with a dry and staring coat; the perspiration gives off an offensive odour.

General weakness soon manifests itself by the weight of the hind quarters being thrown alternately and incessantly from one posterior extremity to the other.

When the animal is caused to walk, the gait is very uncertain, and the sudden flexion of the joints is sometimes so great as to cause it to fall. There is a considerable tumefaction around the larger joints, and especially around that of the haunch. This tumefaction is very painful, and causes the animal while at rest to lift its leg with a sudden jerk, after the manner in which a horse with stringhalt behaves when caused to move suddenly, but it places it more carefully on the ground again than does the latter. When both limbs are affected, this movement is manifested alternately. These symptoms of lameness are sometimes intermittent, disappearing for a week or two, and then reappearing with a greater intensity.

After some time the appetite becomes exceedingly capricious, but is never entirely lost, the animal continuing in many cases to eat till within an hour of its death.

One or both hind extremities now become paralysed, causing the sufferer to fall upon its litter, and the entire muscular system becomes atrophied, but more especially the hind quarters and legs.

The mucous membrane of the nose becomes of a yellow and the buccal membrane of a leaden colour; and there may be slight constipation.

Unless pneumonia or other complications of the respiratory organs set in, the respiration remains natural. M. Rodloff has often observed horses suffering from this disease become broken-winded.

In the mare, one or other half of the mammary gland sometimes becomes the seat of an inflammation, which usually terminates in suppuration.

*Post mortem* reveals great emaciation, no fat, blood pale and small in quantity; yellow, gelatinous exudation beneath the skin, and in dependent parts of the body; enlarged spleen; bones fragile (from removal of salts); bone marrow of a brownish hue; articular cartilages often eroded.

The cord in the lumbar region is reddened and softened, and the superior columns degenerated.



*Treatment.*—Castrate the stallions and segregate all mares. In a country like Britain we should undoubtedly destroy all affected and suspected animals. As a matter of fact, once an animal contracts the disease there is little or no hope of curing it, and it is always a source of danger to other animals, so the above course is much the best for all concerned.

#### CONTAGION.

The contagious properties of the disease have been denied by some, but the experiments of M. Lafosse are sufficient to convince the most incredulous. In 1852 that gentleman took fifteen healthy mares from a regiment stationed at Toulouse (where the disease had never been seen, and where it has never since reappeared), and presented them to affected stallions which had been brought for the purpose from the Dépôts de Tarbes, where the disease was then raging. Five of these mares became affected to a slight degree, the disease disappearing spontaneously. Five others became seriously affected, one of which only recovered; thus showing that five only entirely resisted, while four succumbed to the disease.

Other experiments could be related, but I think the above is quite sufficient to show that its transmission during the act of copulation is undeniable.

It is, however, just to observe that numerous experiments by inoculation were conducted without result, before the contagion was denied.

M. Hering, notwithstanding, succeeded in one instance in producing the disease by rubbing the mucous membrane of the vagina with the virulent matter.

It is worthy of remark that stallions offer more resistance to the contagion than mares.—(M. REYNAL.)

The actual cause, as already stated, is the *Trypanosoma equiperdum*. The organism is never found in great numbers. In the early stages it may be seen if a very careful examination be made in the discharge from the local lesions, but more certainly by harpooning the testicle and obtaining some of the seminal fluid. It is invariably found in the plaques in the second stage, and in the third stage has been seen in the spinal fluid. The presence of one organism is sufficient for

diagnosis, and this may entail the examination of many preparations. It is not so active or so large as the surra parasite, and is no more present at periods of fever than at any other time. It is rarely, or never, found in a droplet of blood taken from the general circulation except when done so from a plaque.

Contagion is entirely by coitus, and not through the medium of any fly. In this it differs from all other trypanosomata.

#### POST MORTEM APPEARANCES.

The *post mortem* appearances with regard to the genital organs do not coincide in different subjects. The lesions of the external organs and of the mucous membrane are, however, constant.

On cutting through the skin it will be noticed that invariably there is a layer of thick yellowish gelatinous exudate, which drips continuously a yellowish fluid. This is observed over the whole of the subcutaneous tissue, but particularly of the abdomen.

The sheath, when cut into, presents the appearance of a hard, homogeneous, yellow tissue, but on a closer examination the fibres of the areolar tissue may be distinguished, and between them the serosity which gives the appearance indicated.

The penis is partly protruded from the sheath, and slightly infiltrated towards its free extremity. On its external surface, though very rarely, small whitish elevations, formed by the infiltration of a small quantity of serum under the epithelium, have been observed. Small dried coagula of blood, which give the ecchymosed appearance mentioned in the symptoms, have also been encountered; but the mucous membrane offers no other alterations than a slight protrusion, a thickening, and a yellowness in colour.

The mucous membrane of the vesiculæ seminales is said by some to offer a red or violet appearance, containing a yellowish thick matter of a purulent aspect; by others, the vesiculæ seminales, as well as Cowper's and the prostate glands, are said to be in a normal condition.

The testicles may either be healthy, atrophied, or hypertro-

phied. When hypertrophied, a yellowish serosity is found in their interior. In the mare the mucous membrane of the uterus and vagina is thickened, ecchymosed, and of a reddish-brown colour. In the uterus a muco-purulent, yellowish-white, or a chocolate-coloured fluid, analogous to that which flows from the vagina during the course of the disease, is found in greater or less quantity. It is said to have been so abundant in some cases as to have given the patient the appearance of being pretty far advanced in gestation.

The kidneys may be either in the normal condition or they may be hypertrophied. Their pelves contain a substance resembling the white of an egg, which has been found to be abundant in albumen. M. Lafosse has found them to contain, in exceptional cases, a substance similar in appearance and consistence to honey.

The mucous membrane of the ureters and bladder of both sexes present a similar aspect to that of the womb in the female.

The muscles are friable, as are also the bones, especially the femur and ribs, of which the spongy tissue is generally impregnated with a dark-coloured blood, or with a yellow gelatinous matter.

The coxo-femoral and pubio-femoral ligaments are red, thickened, softened, and sometimes ruptured.

The synovia of the articulations is abundant, muddy, and presents the colour of blood.

The cartilages are yellow and soft, and have sometimes disappeared from the articular surfaces.

The fatty matter has everywhere disappeared.

The subglossal, sublumbar, and mesenteric ganglia, as also those in the vicinity of the urino-genital organs, are considerably enlarged, and of a yellow or reddish-brown colour. They are sometimes found to contain purulent matter. The French authors describe no other lesions in the cranial cavity than the superabundance of the subarachnoidean fluid, while the German authors affirm that the brain is in a degenerated state. They, however, agree that in some cases the spinal cord is softened, particularly in the lumbar region. It may, however, be remarked that when the disease terminates in paraplegia, if this condition of the cord be not found, it is at least slightly congested.

Occasionally the volume of the sacro-lumbar plexus and sciatic nerves is augmented by a serous infiltration.

The digestive organs are healthy; in some instances, however, the liver and spleen are enlarged and softened.

The lungs are healthy (unless the disease has become complicated by pulmonary affections), but the mucous membrane of the bronchial tubes and larynx is slightly affected.

The sinuses of the head often contain a yellowish, oily matter, and the Schneiderian membrane is of a leaden colour, with red spots scattered over its surface.

The muscular tissue of the heart, like the rest of the muscles, is soft, friable, and of a pale yellow colour.

The blood is very much altered; it is fluid, deficient in fibrin, and, according to M. Lafosse, who has made microscopical examinations of it, the coloured corpuscles present a deformed appearance.

#### TREATMENT.

Antiphlogistics (including bleeding), emollients, tonics, stimulants, alteratives, &c., have all been employed in this disease, but with so little success, that until the appearance (in 1865) of M. Trelut's memoir it was believed incurable.

M. Trelut, after a careful study of the disorders produced in the organism by the *maladie du cort*, adopted an entirely different treatment, the efficacy of which is beyond a doubt.

Being struck by the fact that the blood of animals suffering from the disease was deficient in one of its most important elements, viz., the fibrin, he thought that if this constituent were renewed in the vital fluid the disease might be overcome.

He accordingly procured the necessary fibrin by stirring the blood of cattle when warm, thus separating the fibrin from the other constituents. The fibrin obtained in this manner was dried in an iron pot, in which a little butter had been melted to prevent carbonization; it was then divided into small particles and administered in the morning, fasting, in doses of sixty grammes (̄xv.), either as an electuary, or added to half a quart of gruel as a drench. To stimulate digestion twenty or thirty grammes (̄v. or ̄vii ss.) of turpentine were administered every second morning with the drench. From ̄i. to ̄ii. ss. of iron, reduced by hydrogen, alternated with from

fifteen to thirty grains of the white oxide of arsenic, were also given, and food of the most substantial kind was allowed.

As the patients under the charge of M. Trelut increased, he found that a sufficient quantity of fibrin could not be obtained in the manner described. He then substituted for the pure fibrin of the blood of cattle cooked horse flesh, very finely divided, and administered in the water which had served to cook it. When the bouillon was finished, the flesh was mixed with honey and given as an electuary. Were it not important to utilise the soup, says M. Trelut, the flesh could always be given most conveniently in the latter manner.

The flesh was administered in doses of from 100 to 150 grammes.

When paralysis set in, cantharidine liniments, followed by mustard poultices, were applied to the abdomen; and as soon as an abundant effusion had been established, the pointed firing-iron was applied, penetrating as deeply as possible, in order to fix the engorgement. After the establishment of this engorgement the paralysis became gradually ameliorated, and in twenty-four hours, the animal, which was helplessly stretched upon its litter, was able to regain its feet without assistance.

The internal treatment requires to be continued at least forty-five and at most 145 days, and on an average from two to three months.

By this treatment M. Trelut was successful in sixteen cases (in which the patients were seriously affected) out of seventeen.

The seventeenth was a mare which aborted, and the lesions of metro-peritonitis found at the autopsy would have been sufficient to account for her death.—(*Memoire sur la Maladie dite du Coït*, par M. TRELUT, *Recueil de Médecine Vétérinaire*, Janvier, 1865.)

*Local treatment.*—In stallions, during the first stage, or that in which the disease is confined to the genital organs, it was stated that good results had been obtained from castration.

In the mare, mucilaginous injections, followed, later on, by mild antiseptics, astringents, and still later, if necessary, by the sulphate of copper, sulphate of zinc, or nitrate of silver, &c., have been recommended.

Castration is beneficial in the early stage, and probably acts by removing all the organisms, as they would appear to

be located in this region for a considerable time ; it is useless after the disease has become generalised. No satisfactory medicinal treatment has yet been discovered, although it is quite certain that cases do recover without any treatment. It is probable that the alternate administration of arsenic and mercury, as it is now being applied in the treatment of sleeping sickness and nagana, and was first suggested by Lingard fifteen years ago, might have beneficial results. The theory of giving the two alternately is that the arsenic acts upon the organisms when they are motile in the blood-stream, but does not kill them ; they simply become encysted. The mercury will then attack them and cause their speedy dissolution.

#### COITAL EXANTHEMA OF HORNED CATTLE.

It is stated in Friedberger and Fröhner's works on pathology and therapeutics of the domestic animals that this disease is more frequent in cattle than in horses simply on account of their greater number.

It is very contagious, as all the cows of a village may be infected by one bull, and it is said that it is communicated by friction of diseased against healthy cows, and by continual wagging of the tail. In oxen Numann has observed the eruption around the anus and sheath. Armbruster states that the contagion is conveyed by wooden drains—when animals lie down, their genitals coming in contact with these drains—when these are contaminated by diseased discharges. Schnepfer says that the disease may affect the same subject several times at short intervals.

The symptoms are similar to those observed in the mare. There is intense pruritus, vaginitis with redness and tumefaction, muco-albuminous secretion ; membrane covered with dark red spots, vesicles, and pustules of various sizes, which rapidly ulcerate, the discharge from which contaminates the perineal region and tail, drying up, forming crusts ; micturition is difficult, and manipulation causes much pain. The appetite, secretion of milk, and other signs of general disturbance are frequently present. The duration of the disease seems to vary from eight to fifteen days, and more rarely from three to four

weeks. Some cows abort. In the bull there is much tumefaction of the genitals, the penis studded with pimples, and there is a discharge of a yellowish purulent material from the urethra; phymosis is frequent; erection causes the ulcers to bleed; there is more or less constipation and loss of appetite.

Local antiseptic and astringent treatment is recommended.

### ANÆMIA—SURRA.

A fatal form of anæmia has been observed on the Continent of Europe, in India, and Burmah. Some observers state that it is due to a fine bacillus in the blood, and that it is infectious; whilst Evans, Burke, Steel, and Lingard, who have studied it in India, where it is well-known under the name of SURRA, have discovered that it is due to the presence of hæmatozoa—the *Trypanosoma* (see Chart)—in the blood of the affected animals—horses, mules, camels, elephants, donkeys. The hæmatozoa are also found in rats and bandicoots, and in honour of its discoverer, Evans, it is now called *Trypanosoma Evansi*.

Evans, when he first discovered the parasite, thought it was a spirillum, but further examination convinced him that it belonged to the animal kingdom, presenting when fresh and active an apparently round body, tapering in front to form a neck, and terminating in a blunt head, and posteriorly a tapering tail, extending from which is a long slender lash. He also states that at the head end there appeared in one or two cases a circlet of pseudopods, and as the body slowly died in serum it gave the appearance of flattening out. After watching carefully all the forms and movements of the parasite, he concluded that on each side of the body there existed two pin-like papillæ, one near the neck and the other close to where the tail began; but he only observed them in a very few instances, and supposed they were of the nature of pseudopods.

The parasite is very active in its movements, with an undulatory eel-like motion, moving generally with head foremost, but occasionally in the direction of the tail lash, when, as Captain Appleton, A.V.D., describes it, tugging at and worrying a red corpuscle: they thus attack and disintegrate the red corpuscles.

Sometimes two will unite and swim off as one body. Evans thought that they joined heads and tails; others thought that they fastened with their tails in opposite directions.

The disease is not contagious or infectious in the ordinary sense, but can be communicated by subcutaneous inoculation, and by the introduction of blood containing the parasites into the stomachs of healthy animals.

Steel differed from Evans as to the exact classification of the parasite, maintaining that it resembled the spirillum of relapsing fever in man. Notwithstanding this error, it must be admitted that he did good work when the disease broke out in British Burmah in 1885, confirming its communicability by inoculation of blood containing the parasites to the dog, horse, mule, &c., that the disease was relapsing in its nature, that the parasite appeared as the temperature rose, and disappeared during the non-febrile periods; and he concluded as follows:—"That relapsing fever of mules is invariably a fatal disorder, characterised by the periodical occurrence of attacks of high fever, during which a special organism closely resembling the spirillum of relapsing fever in man is found in the blood. This organism is one-sixth of the size of a blood corpuscle in width, and three to six times in length, &c. All observers conclude that the parasites are not always present in the blood of the diseased animal, but come and go in successive broods."

Seeing that Evans and Steel differed in opinion as to the exact classification of the surra organism, Evans referred the matter to the late Dr. Timothy Lewis, who concluded that it resembled, but was not exactly like, that which he had discovered in the blood of rats, now called *Trypanosoma Lewisi*. The matter was further referred to Crookshank.—("Journal of Royal Microscopical Society," part 6.—"Flagellated Protozoa in the blood of diseased and apparently healthy animals.") He says, page 916:—"In the face of these conflicting opinions, Dr. Evans was good enough to place in my hands for investigation some preparations of the organism in the blood as well as material from the lungs and intestines of a camel that had succumbed to the disease.

"On examining a stained preparation with a power of 200 diameters, a number of the parasites could be distinguished in the field of the microscope, and with a 1/12 and 1/18 O.I. (oil



immersion) the individual characters were clearly brought out. These were quite sufficient at once to dispel the idea of its being a spirillum. It was obvious that it was a more highly organised micro-parasite, presenting very peculiar and distinctive structural appearances.

"The first glance at the parasites recalled the appearance of nematode hæmatozoa, as if, indeed, they might be embryonic *Filariae*; but when I had carefully studied several specimens, and had further undergone the searching examination entailed by the accurate focussing necessary to obtain a number of sharply defined photo-micrographs, I came to the following conclusions:

"The somewhat tapering central portion or body of the parasite is continuous at one end with the whip-like lash, and at the other end terminates in an acutely pointed stiff filament or spine-like process. Here and there, possibly from injury or want of development, the spine-like process appears to be blunted or absent. By carefully focussing on the upper edge of the central portion, I discovered the existence, much more markedly in some of the parasites than in others, of a *longitudinal membrane*, with either straight or undulating margin. The membrane is attached along the body, arising from the base of the rigid filament, and becomes directly continuous at the opposite end with the flagellum. In some cases the edge only is deeply stained, giving the appearance of a *thread continuous with the flagellum*, so that one might be easily led to overlook the membrane, and imagine that the flagellum arose from the opposite end of the body, at the base of the spine-like process.

"Close to the base of the spine-like process, a clear unstained spot is in many parasites easily distinguished, and at the opposite end there is in some the appearance of the deeply stained protoplasmic contents having contracted within the faintly stained membranous investment. Where the longitudinal membrane has a wavy outline the undulations are much more marked in some cases than in others. Here and there the wavy outline appears first on the one side of the central portion and then on the other, but there never is any wavy outline on both sides of the same part of the body; and this was explained by careful examination, which showed that in dying the somewhat ribbon-like parasite had become doubled on itself. The discovery of this undulating membrane at once suggested to my

mind an explanation of the lateral pseudopodia described by Evans.

"If we imagine that we are looking down upon the parasite, with the edge of the membrane towards us, one can conceive that the rapid undulations, first on one side and then on the other, might give an image upon the retina which could be construed as due to protrusion of lateral pseudopodia.

"I may add that I could not discover in the stained preparations any trace of the circlet of pseudopods, and I think the undulating membrane may account for this appearance also."

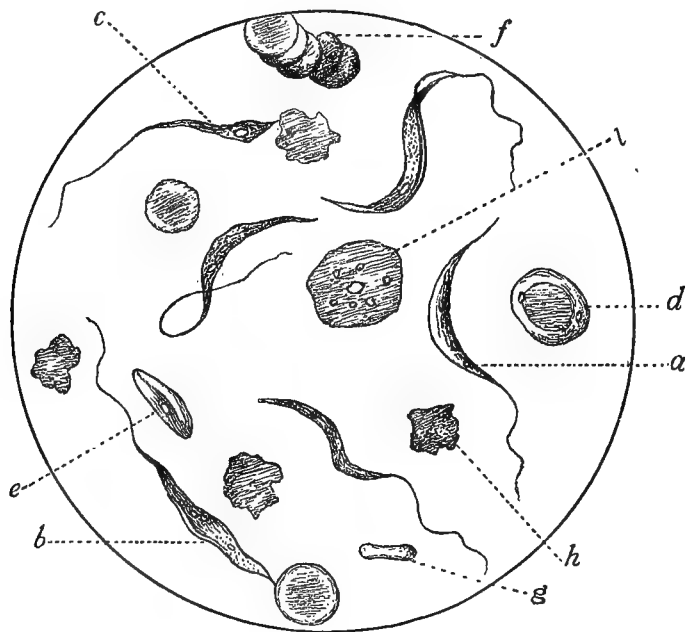


FIG. 39.—Monads in Rat's Blood.

- a. A monad in free motion among the cells.
- b. Another, attached to a red corpuscle.
- c. Angular form.
- d. Encysted form.
- e. The same seen edgewise.
- f and g. Normal red blood corpuscles.
- h. Crenated red blood corpuscle.
- i. White blood corpuscle,

Owing to the somewhat curved and twisted shape of the parasite, and the curling of the flagellum, in the stained preparations it was difficult to make exact measurements, but I was able to ascertain that the average width, according to whether the membrane was visible or not, varied from 1 to 2  $\mu$ , and the length of the body from 20 to 30  $\mu$ . The flagellum was about the same length as the body.

Similar, if not identical, parasites are found in the blood of rats, bandicoots, mudfish, and carp, apparently inducing no inconvenience to their hosts. There is no longer any doubt that the *Trypanosoma Evansi* is the causal agent in surra. This protozoan is of the family Flagellata, measures from 25 to 30  $\mu$  long, and is from 1 to 5  $\mu$  broad. The flagellum, a continuation of the undulatory membrane, is at the anterior end. It usually moves in this direction, but may proceed backwards. There is an ovoid granular macro-nucleus about the centre of the body, and a small circular micro-nucleus or centrosome, composed of chromatin at the posterior extremity, from which the flagellating membrane arises; this membrane is also composed of chromatin. Multiplication occurs by longitudinal division, usually commencing at the centrosome.

It is extremely probable that there is an intermediary stage occurring in some other host, in the same way that the malarial parasite of man has an intermediary stage in the mosquito. At present our knowledge as to the habitat of these organisms during the apyrexial stage of the disease is very incomplete, although encysted forms have been observed in the spleen. This subject is at present engaging the attention of many scientists. In the case of nagana, the means by which the disease is conveyed is by a biting fly, named the *Glossina morsitans*. The method of contagion is purely mechanical—i.e., a fly bites an infected animal, and some organisms collect with the indrawn blood in the proboscis of the fly. This dipterous insect, by then biting another healthy animal, will infect it with living trypanosomes. In the case of surra, a fly has not been absolutely demonstrated as the carrier, but there is considerable evidence to show that a fly of the family Tabanidæ is the agent that transmits the disease from infected to healthy animals.

The question as to the method by which the disease is

carried over from one fly season to another is a difficult one; but it would appear from investigations made by Pease and others that cattle may be infected with the disease although they show no pathogenic symptoms. These animals are bitten by the *Tabanus*, indiscriminately with horses, and may serve as a medium for promulgating the infection. The whole subject, however, requires further experimental research. The question of rats conveying infection is not now taken into consideration, the parasite usually found in these animals being the *Trypanosoma Lewisi*, which has nothing to do with surra, though the parasites are much alike. Rats may acquire surra, but probably only by experimental inoculation. Corn, grain, and food generally may also be eliminated, and we may now accept that surra is entirely a fly-borne disease. The rat, owing to its habits of life, being very unlikely to suffer from the attacks of *Tabanidæ*.

It was discovered by Lingard that one attack did not render an animal immune to a second, and the following experiment is interesting as bearing on this point: Twelve months after an Australian horse had been cured of surra by the administration of arsenic, iodide of arsenic, and mercury, this horse was inoculated with a minute drop of surra blood—a mere trace of soiled blood being smeared over a scratch on the muzzle—in order to ascertain whether any immunity had been given by the previous attack. The latent period of the second attack occupied seven days. The progress of the disease was marked as usual by paroxysms of fever and intermissions, the former occupying a much more prolonged period (twenty-two days) and the latter being shorter than usual (only two days).

Death took place on the fifty-third day of the disease, clearly proving that one attack of surra is unable to protect against a second attack.—(Lingard, *Report on Surra to the Inspector-General, Civil Veterinary Department, Simla*, 1895.)

*Symptoms of Surra.*—Outbreaks of this disease occur in India and Burmah chiefly after the rainy season. The more prominent symptoms observed at first are a capricious appetite, dulness, and slight fever, which after a few days' rest frequently pass off without medical treatment. When a relapse occurs, which is almost invariable, there is an increase of fever, the mucous

membranes are yellow in colour, and petechiæ become apparent, also local or general urticaria. There is generally some œdema of the limbs, also beneath the belly, chest, and sheath. The urine becomes much altered, viscid, of a dark yellow or orange-green colour, frequently containing albumen and blood. The animal becomes emaciated, and great thirst is invariably present. There are occasionally catarrhal symptoms. The lungs may be more or less congested, and breath fœtid. There are periods of remission, marked by improved health, which is merely transitory, as repeated attacks of increasing intensity finally end the animal's life. This disease, according to the condition and age of the animal attacked, may last from a few days to several months before death occurs. It is very fatal. The disease, until quite recently, has not been amenable to any line of medical treatment. Professor Lingard, however, has been experimenting with the arsenic treatment, with some amount of success in patients possessed of vigorous constitutions.

### NAGANA OR TSETSE-FLY DISEASE.

Due to the injection into the circulation of horses, cattle, dogs, goats, donkeys, and wild animals of trypanosomes (*Trypanosoma Brucei*), in one of their stages of development, by the tsetse-fly.

#### *Symptoms in the Horse.*

The period of incubation is from fifteen to twenty days.

After this period of incubation, during which the hæmatozoa multiply to an enormous extent, we notice that the patient is dull, his coat stares, and there is a watery discharge from his eyes and nose. Shortly afterwards swellings appear beneath the abdomen and in the sheath; then the hind-legs become swollen—these swellings are remarkable in the fact that they come and go; during this period he rapidly loses condition and becomes weaker and weaker each day, even though the appetite is maintained till death supervenes.

The mucous membranes at first are pale, the cornea be-

comes cloudy, and he may become totally blind, and the discharge from the eyes continues to increase.

In the later stages the conjunctival mucous membrane is covered with dark purple petechiæ and patches.

During the progress of the disease the temperature is very variable, somewhat as follows on consecutive days—98°, 103·5°, 100°, 106°, 102°, 105°, 100°, 107°, 100°, 102° F., and so on for about six or seven weeks, the afternoon temperature being as a rule higher than that of the morning, the patient by now being nothing but a bag of bones—so thin in his quarters that his anus falls inwards and his thighs are quite apart—his coat is ragged, dirty, scurfy, and the hair has fallen off in many places, and there are dropsical swellings at the dependent parts of the body. He becomes too weak to stand, and ultimately dies of exhaustion.

If during the course of the disease an examination of the blood be made, the trypanosomes can be easily seen, varying in number on different days, some days great in number—and on such days temperature is high—other days *vice versa*. Blood examination also reveals that the number of red blood corpuscles is greatly diminished. Whilst in America in 1900 I saw a considerable number of such cases in Texan ponies, but had no opportunity of proving it by microscopic examination of the blood.

### MAL DE CADERAS.

This is a specific blood disease due to the presence of trypanosomes, named the *Trypanosoma equinum*, which is stated by Vosges to be transmitted from animal to animal by a fly belonging to the species *Musca brava*.

The disease is specially seen in Central America, and is called *mal de caderas*, or disease of the hind-quarters, on account of one of its most marked symptoms.

*Asses and mules* are not so susceptible as the horse.

*Guinea-pigs* are even less susceptible, but *rats and mice* die of the disease very quickly.

*Dogs* die in two or three months.

*Sheep and goats* contract the disease, but may live for months, and then they quickly become emaciated and die.

*Birds* die of it in two or three weeks.

*Cattle* appear to be immune from this disease.

There has been considerable confusion amongst observers as to the differentiation between *dourine*, *surra* and *mal de caderas*, but it is now generally agreed that they are three distinct diseases, in so far as the causal agents are concerned, though at the same time many of the symptoms are common to each of the three.

In experimental cases, Vosges says, if 5 to 20 c.c. of blood from an affected horse be injected into a healthy horse, a fever develops in four or five days, and increases in severity *hourly* until the following morning, the temperature going up to as high as 40° C. to 41° C., or about 104° F. to 106° F., and then during the day falling to normal or almost so, and reaching its minimum on the second day of the fever.

The temperature again commences to rise, and gradually rises for four or five days until it attains its maximum, about 105° F. This periodic rise and fall may recur four, five or even more times, with intermissions of three days to a week.

During all this the pulse and respiration remain normal, and the appetite is only slightly diminished, and that occurs in the evenings.

The thirst, however, is increased.

Sometimes the fæces are streaked with blood.

Hæmo-albuminuria is observed and remains constant.

The coat retains its healthy gloss and is cast at the proper time of the year.

In the second stage, which *gradually* replaces the first, the intermittency of the fever disappears, the temperature does not so markedly rise and fall, but fluctuates round about 101° F. to 104° F. The heart becomes weak, the animal droops, loses condition rapidly, and is always thirsty. The coat stares, œdematous swellings appear in the hind-legs, and later, in other dependent parts of the body. The skin becomes anæsthetic, and flies on it seem to cause no irritation or annoyance. The urine is abundant and always contains blood.

The animal staggers when moved, and seems partially paralysed behind, and may fall, and after some struggling will die. Some may remain standing until the last moment and then drop dead, without any pain or suffering.

Towards the end, the morning temperature may be as low as 34° C. or 93·2° F., and in the evening rise to 39° C. or 102° F.

The disease runs a course of from fourteen days to four months, and is only seen in swampy districts.

If horses be stabled at night, they do not become affected, and if the disease break out in a large number of horses they should at once be removed to a high and dry locality.

### *Post Mortem Appearances.*

Œdematous swellings in various parts of the body; coat harsh and staring, body much emaciated, hide difficult to remove, muscles dry, as seen in cholera patients; pleural cavity contains large quantity of clear or almost clear yellow serosity; pericardium filled with similar fluid; heart pale and flabby.

The abdominal cavity contains a large quantity of fluid similar to that in the thorax.

The liver is enlarged, so are the kidneys. The *spleen is enormously enlarged*, the enlargement being in proportion to the duration of the disease, and the various lymphatic glands are all swollen.

*The blood-count* shows an enormous decrease in the number of red blood corpuscles from 10,000,000 down to 3,000,000, or even 800,000; at the same time there is an increase in the number of white blood corpuscles.

The parasites appear in the blood in five days after inoculation; they increase in number as the temperature of the patient rises, but suddenly disappear when that temperature is up to 40° C. or 104° F.

In about three or four days they again appear and increase in number as before, and disappear when temperature is again 104° F.

When they are most numerous, they may be in the proportion of 1 in 10 to 1 in 4 of the blood corpuscles.

The blood loses its infectivity in from twenty-four hours to two days after death. So far all medicinal treatment has been unsuccessful.



## CHAPTER XLIV.

### BLOOD DISEASES—*continued*.

#### PIROPLASMOSIS.

PIROPLASMOSIS (*P. bigeminum*) is a disease due to the presence of piroplasmata in the blood, and more particularly in the red blood corpuscles, where these parasites exert a malignant influence, and cause a breaking down of the corpuscles. These parasites are introduced into the economy by ticks of various species, and give rise to the following well-recognised forms of disease: Texas Fever, Jamaican Fever, Malignant Jaundice in the dog, Biliary Fever in the horse, African East Coast Fever, Red Water, and Black Water.

#### TEXAS FEVER (IXODIC ANÆMIA).

Later studies of this disease have brought to light that it assumes two forms: an acute fatal type, as seen in America in the hot summer months, and a mild, rather prolonged, usually non-fatal form, recognised by an examination of the blood, which must reveal the micro-parasite in the red corpuscles and plasma. This form is seen in the autumn when the heat of summer has passed away, also during October and November, and, rarely, in the first week of December. It is stated that the difference between the acute and mild type is accounted for by the fact that during a stage of its life—the small stage—the Texas fever parasite circulates in the blood in a condition differing from that observed in acute cases; that in the latter it rapidly destroys the blood corpuscles, giving rise to hæmoglobinuria—red water—whereas in the mild form the destruction of the corpuscles is much more prolonged, and not associated with hæmoglobin in the urine.

It has also been observed that in certain cases when an animal has recovered from the acute attack, and the number of red corpuscles has nearly reached the normal number, they again diminish, and many of them contain the parasite in its small stage. The reason for the recurrence of this disease is not clearly defined,—whether these second attacks are merely relapses or reinfections. As already stated, the symptoms of the mild form are indefinite, and may be confused with a variety of mild diseases, but the *acute form* is manifested suddenly in the hot summer months, and simultaneously in all animals of a herd which have been exposed to the same infection together.

The fever, characterised by a temperature of from 105° to 108° F., precedes outward symptoms for several days, the animals apparently quite well. The pulse and respiration then become accelerated, and the urine is now observed to have the appearance of blood, varying in colour,—reddish, claret, or blackish red water (hæmoglobinuria) in the most acutely fatal cases. Out of forty-six fatal cases (Bureau of Animal Industry, Washington, 1893), hæmoglobin was found in the urine in the bladder of thirty-three after death. It is uncertain whether this condition of the urine is present in all cases of the acute form, the opinion of the reporters to the American Bureau being that it “depends upon the rapidity with which the red corpuscles are infected and destroyed. A slow destruction may allow other organs to excrete the *débris* and thus forestal the discharge of hæmoglobin in the urine.” But if free from hæmoglobin, the urine at the height of the fever is found to contain a small quantity of albumen, to have a specific gravity of 1030-40, may be strongly alkaline and effervescent when treated with acids; but as the disease advances its specific gravity will fall to 1010-12, its reaction becoming neutral or slightly acid. It sometimes contains small numbers of red corpuscles, which may be derived from small hæmorrhages in the pelvis of the kidneys, regularly observed *post mortem*.

The bowels are, as a rule, constipated during the high fever, but as the fever subsides the fæces become softer and tinged with bile. After a few days' illness the debility becomes very great, and whilst the animal is standing, trembling of the hind quarters and limbs are prominent features in many cases: the blood is then found to have become very thin, pale, and watery,

due to the destruction of the red corpuscles. In the earlier stages, if freshly drawn blood be allowed to stand, the serum will be found to present a very dark red colour, indicating the presence of much colouring matter in solution. Later on, this colour may not be present, but thinness of the blood, owing to destruction of the red corpuscles, seems to be the "most essential character of Texas fever."

The duration of the disease varies, proving fatal or disappearing in a few days. Recovery, however, is associated with great debility; degenerations may occur in organs, and frequently there are relapses. Some animals never regain their health; in others, recovery takes place after weeks or months. High temperature rarely lasts longer than eight to ten days; it is then followed by a period of normal or subnormal temperature, the falling of the temperature marking the cessation of the destruction of the corpuscles, and the disappearance of the parasite from the blood. The mortality varies greatly. A mid-summer outbreak, when acute in its nature, is the most fatal, and from this time there may be all gradations towards the mild, non-fatal form of the disease.

*Etiology.*—Several observers have reported the discovery of various forms of bacteria in this disease. In 1883 Salmon described a diplococcus obtained from spleen cultures. Detmers found bacilli and micrococci in the liver, but none in the blood; and in 1888 Billings claimed the discovery of what he terms the "true germ," an ovoid bacterium, staining at the ends, similar to that of swine plague. All these contentions, however, have now been apparently terminated by the discovery of a parasite in the blood corpuscles, conveyed from Southern to Northern cattle by cattle ticks (*Boophilus bovis*). At the experimental station of the Bureau, near Washington, it was found by experiments in 1889 that the disease can be produced by ticks artificially hatched in the laboratory.

The parasites thus conveyed are found in certain proportions of the red corpuscles in the form of rounded or somewhat ovoid or pyriform bodies, isolated or in pairs, in the acute form of the disease, whilst in the mild form it appears that from 5 to 50 per cent. of red corpuscles are found to contain coccus-like bodies, sometimes on the border, and outside the corpuscles; they are from  $\frac{1}{27000}$  to  $\frac{1}{50000}$  of an inch in diameter, sometimes dividing

into two ; they, as well as the larger pyriform bodies seen in the acute form, stain with aqueous solutions of aniline dyes, and with hæmotoxylin. Both the large and small forms are considered to be different stages of the same parasitic protozoa (*Pyosoma bigeminum*, Dr. Theobald Smith). The smaller ones are found in cattle exposed to the disease late in the season, or during a second attack or relapse after passing through an acute attack, and in milder cases during or previous to the season (hot) of the acute disease.

With the better acquaintance of these intra-corpuseular parasites, improved staining methods have been acquired. The way now adopted is that first recommended by Romanowsky, or a modification of his method. It is based on the principle that methylene blue as a basic dye will stain the protoplasm of protozoans, and eosin will stain the chromatin of the nucleus, the flagellum, and centrosome. The methylene blue and eosin are prepared by special methods, and used as a mixture or separately in definite proportions. By this means very beautiful specimens are obtained.

The blood-film is first fixed in alcohol after rapidly drying in the air. The mixed stain is then applied for a definite time, and the reaction will be to stain the red corpuscles pink, and the parasite within them a blue, and within the parasite will be seen a small red mass of chromatin.

*Post Mortem Appearances.*—Congestion of the vessels and occasional patches of extravasation in the subcutaneous tissues, blood sometimes thin and watery, lungs normal or discoloured by congestive patches, heart congested ; petechial spots on peri- and endocardium, spleen congested, its capsule streaked from distension of its vessels. The weight of the organ varies, according to the stage of the disease in which the animal dies, the more rapid the fatal termination the larger the spleen. Generally it is from two to four times its natural weight, distended, firm to the touch, its capsule attenuated, the pulp dark brown, glistening, and homogeneous. The markings of the Malpighian bodies and of the trabeculæ have disappeared.

*The liver* is gravely altered, enlarged, sometimes of a dark colour from blood congestion, or light coloured from extreme bile engorgement ; gall bladder full of thick grumous bile ; when

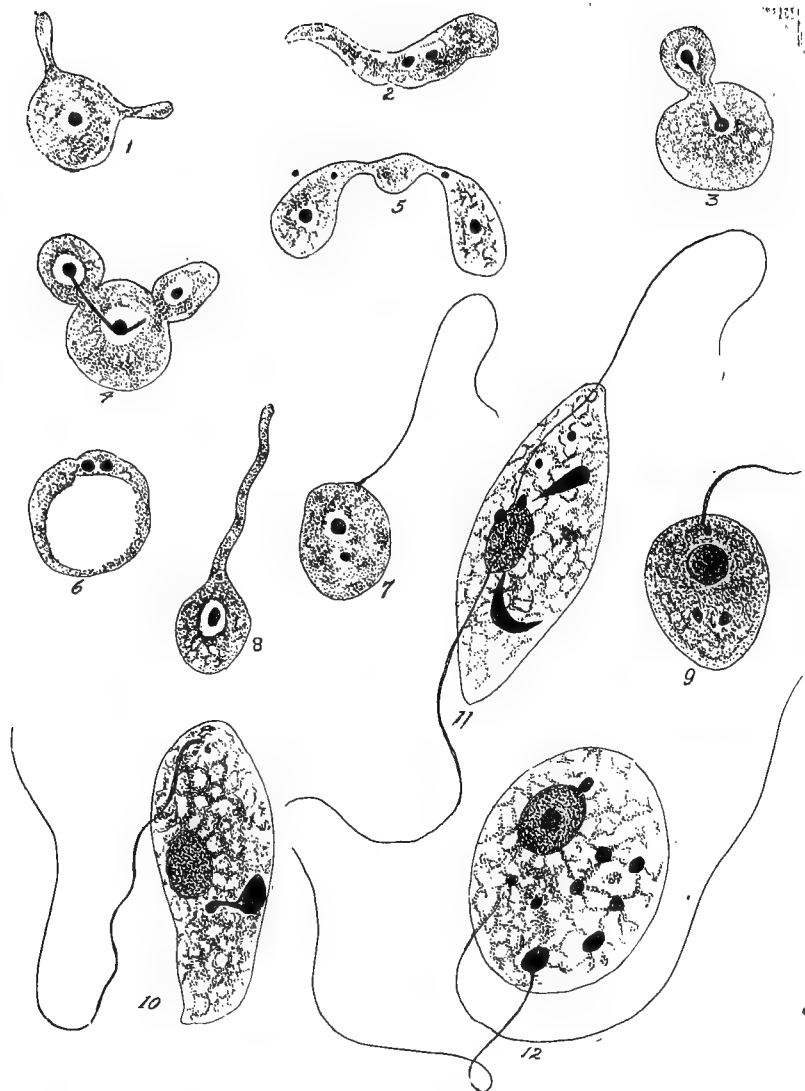


FIG. 40.—Various phases in the development of *Piroplasma* of the dog.

(From article by Drs. Anton Breinl and Edward Hindle, in the *Annals of Tropical Medicine and Parasitology*, University of Liverpool, July 1st, 1908.)

1 and 2, Early forms of parasite ; 3, single budding ; 4, double budding ; 5, pear-shaped form ; 6, signet-ring form ; 7, free small flagellate parasite ; 8, amoeboid form with long pseudopodium ; 9, formation of flagellum ; 10, extrusion of chromatin from the nucleus ; 11, extrusion of two masses of chromatin ; 12, development of large bi-flagellate forms with characteristic nuclei.

incised the cut surface presents a uniformly brownish-yellow colour, or else a mottled appearance, also seen on the surface. There is occlusion of the biliary canaliculi and ducts, and a more or less extensive fatty degeneration of the liver cells, and a tendency to necrosis of the inner zone of the lobules.

*The kidneys* vary in colour according to the severity and stage of the disease when death occurs. In the early stages, when the bladder contains port-wine coloured urine, they are enlarged and of a uniform dark brownish-red colour, and the vessels are uniformly distended with red corpuscles. The secreting structures are not seemingly altered. In cases which die after the acute stage and fever have passed away, they are paler than normal, and quite flabby, having pigmentary deposits in the convoluted tubules, and their epithelium so loaded with a reddish-yellow pigment that they are easily traceable in their windings by their colour. In those cases where the capillaries are filled with red corpuscles, the microbes are usually found in all the corpuscles. Extravasations of blood are found in the pelves, and in most cases the bladder is found to contain urine mixed with hæmoglobin, whilst ecchymosed spots are seen on the inner surface of the mucosa.

*The digestive apparatus*, with the exception of the abomasum, which is frequently congested, generally presents no definite alterations. The pyloric portion of the abomasum is found to contain deep ragged excavations with hæmorrhagic bases. The constancy of these ulcers, probably the result of vascular occlusion, is considered by Moreau Morris to be a more certain indication of Texas fever than any other lesions commonly present.

The intestinal lesions are congestions and pigmentation, with an increased flow of bile.

*Prevention and Treatment.*—If the disease is suspected, the herd should be examined thoroughly for ticks, and the animals' temperature taken. *The combination of ticks and fever, or the presence of the former, in a locality where they do not naturally exist, may be considered a sure sign of the imminence of Texas fever.* If the ticks are found, they should be carefully removed and the cattle at once transferred to uninfected grounds. The examination for ticks should be done repeatedly, and all ticks destroyed. These measures may not prevent all attacks, nor

cut short the disease after it has once shown itself, but the reporters are satisfied that fewer animals will succumb. A single infection is sufficient to cause severe and prolonged disease, as shown by the experimental injection of blood; but the mortality seems to be lower than in natural exposures, where the infection is intensified with every additional tick.

No special line of medical treatment has as yet been tried. Quinine and its preparations are proposed as possibly valuable, and methylene blue, recommended for malaria, is referred to as worthy of trial.

Inoculation with the blood of Southern cattle, apparently healthy, and with that of diseased Northern cattle, transmits the disease, and if this is done after the hot summer weather has passed, the induced disease may be of a mild character; but it appears that immunity is not assured, as it is reported by Dr. Dinwiddie that of vaccinated and non vaccinated cattle sent to Texas  $66\frac{2}{3}$  per cent. of the vaccinated and  $88\frac{2}{3}$  of the non-vaccinated died.

Had there been a well-marked immunity conferred by inoculation, it would have been possible to have improved the method of procedure; and perhaps carefully prepared blood serum, freed from the organism, might have been of value. One remarkable instance is mentioned in the report, namely, that the Texas fever parasite was found in the blood of a North Carolina animal three years after it had left the permanent infected territory.

Similar diseases, manifested by coloured urine, are reported to prevail in the marshy regions of Roumania. These were investigated by Babes in 1888, and disseminated largely by draught oxen: in the Cape of Good Hope also conveyed from place to place by yoke oxen. By keeping communication with the territory north of the colony, these cattle, like those of the Southern States, seem not to infect others directly, but they infect ground over which they have passed. It also prevails in Australia, and is due to the same cause, ticks being numerous in that country.

Infected pastures have been found to be harmless to sheep, and it is thought that other domesticated animals may graze unharmed in such pasture.

The discovery that the disease is conveyed by ticks explains

what has hitherto been very mysterious, namely, that Northern cattle become affected by the disease when pastured on lands previously grazed by Southern cattle apparently in perfect health. This fact leads to the conclusion that Southern cattle, recovered, perhaps, from previous attacks, have acquired immunity, though covered with ticks.

Although it is clearly demonstrated that Southern cattle are dangerous when they bear the cattle tick, and that this tick conveys the micro-parasite from Southern to healthy Northern cattle; but transmission from Northern cattle is very rare.

Northern cattle have ticks on them, but only those which survive the disease, or die after a prolonged attack, ripen the ticks on their bodies. Those which die of an acute attack in a short time after infection have only immature ticks on them. If the fever has occurred early enough in the season to permit a second generation of ticks to appear before the cold weather arrives, another outbreak during the same season may occur. Usually the first outbreak occurs in August, and the second, to be looked for late in September or early in October, is so mild as to pass unobserved. If, however, the first outbreak occur in July, the second may appear in September, and perhaps be of greater virulence.

When the disease is induced by inoculation, experiments have proved that it has no contagious properties,—in fact, that it is not transmitted by cohabitation, but by the intermediation of the cattle tick. But, as already stated, it seems to be concluded that preventive inoculation has so far been a failure, for the effect produced in the body of an animal by the destruction of the red corpuscles equal in amount to all those circulating in the body at any given time should make much more impression than any method of inoculation is likely to do. Not being, so far as at present known, a ptomaine-producing organism, like bacilli, &c., this is as might be expected. Yet such an attack not only does not always prevent a second attack, but may not prevent death during a second attack. Aside from the difficulties attending the production of immunity, under any circumstances the difficulties of preparing a “vaccine” according to the method hitherto practised are at present insurmountable. The micro-organism cannot be cultivated. The infection of Northern cattle might be prevented by careful destruction of the ticks upon



Southern cattle before their removal to Northern territories, or by prohibiting their migration during hot weather. These suggestions are not, however, made by the reporters, but they attach great importance to the protection of animals taken south into permanently infected pastures. They state that it is probable that if calves be taken they may, without treatment of any kind, survive the infection upon southern pastures, and become gradually insusceptible. But in case of animals more than twelve to eighteen months old the first attack might be fatal; and, perhaps, the simplest way of dealing with older cattle is to endeavour to induce immunity by exposing them to infection at some specified time in autumn. In the latitude of Washington it was found that the most convenient time is the middle of September. In more northerly latitudes the exposure should be correspondingly earlier. Cattle exposed in this way invariably take the fever, but the mortality is very small. Such animals may die of a second attack during the succeeding summer, but a mild exposure during the following autumn may furnish a sufficient protection.

Another method of inducing the disease is the injection of blood from infected cattle, which generally induces—if practised after the hot summer weather—mild attacks of the disease. The blood of apparently healthy Southern cattle will serve the same purpose.

Exposure to ticks during the cool autumn months is also recommended, being simpler, requiring no operation, the ticks being easily procurable from the permanently infected Southern territory.

In concluding their observations upon these methods of protection, the reporters say that their statements concerning the possible uses of mild infections as a means of subsequent protection must be regarded as mere suggestions, which may or may not prove of practical utility on a large scale.

In Australia it has been established that blood inoculations from affected animals to non-infected will induce an immunity, but this must be done before such animals are placed upon pastures infested with ticks infected with Texas fever. The inoculation induces an attack of fever which is, however, of short duration and rarely fatal. After recovery the animal is immune, and may be moved with a comparative degree of

safety to infected areas. It is reasonable to suppose that the dose of infection by the inoculation of a slight quantity of infected blood is very small; moreover, the infective material is less virulent than when inoculated directly from the ticks, as well as being so much less than from the bites of numerous ticks. Recovery from this mild attack induces immunity, but it does not follow that the parasites disappear entirely from the bovine's blood, as they may be found for months, or even years, after, and such blood will be capable of infecting ticks, or other animals by inoculation. This explains the danger of allowing apparently healthy animals from infected areas on to healthy clean pastures; even though such animals be quite free from ticks, they may be capable of infecting healthy ticks in the fresh pastures. It is for this reason that every precaution should be taken to eradicate ticks, and this can only be done by systematic dipping or spraying. Without ticks there could be no reproduction of piroplasma, and, without this intermediate host, the disease must in course of time die out.

Appended is a report by Mr. C. P. Lounsbury, Government entomologist, taken from the *Cape Colony Agricultural Journal*:

Tick destruction has become a stock-farming problem of considerable importance in South Africa. Everywhere in the country ticks are more or less of a pest, and in many sections they are a veritable plague. The south-eastern and eastern seaboard is the most afflicted part of the Cape Colony, but stock often suffer severely in southern and south-western districts, and even in portions of the Karroo. Year by year the evil has been increasing, in some places slowly, in others rapidly. A prominent Willowmore farmer recently told the writer that ticks were no trouble at all in his locality five years ago, whilst now the Bont Leg species is so injurious to his stock that he fears he will be driven out of sheep-farming by it.

Various remedies are employed in the Colony to destroy ticks, but most are used in a more or less haphazard manner, with little attempt at system or thoroughness. I am told that many natives practise greasing their cattle, and that at some kraals hogs are reared chiefly to furnish fat for the purpose.

Tick-clipping with scissors, leaving the parasites to dry out on the skin or to drop off as may chance to follow, is a common resort along the coast. One old pioneer of the East London district writes that he pays his native herdsmen to collect female Bont ticks from his cattle, giving them 3d. for each fill of a half-pound jam-tin. In this way he obtained between two and three bushels of ticks last year. A great many farmers now spray or hand-dress with one or another of the numerous carbolic and arsenical sheep-dips at a heavy expense for materials and labour, and unfortunately, through ignorance of the habits of the ticks, most of these men grossly overestimate the value of their applications.

*Incidental Evils from Ticks.*—There are many distinct kinds of ticks on stock in the Colony, and aside from the direct injury the various kinds do in draining off the blood of their hosts, some sorts transmit disease. The common blue tick is no doubt the usual if not the sole agent in the spread of redwater; the Bont tick spreads heart-water; and the dog tick spreads malignant jaundice, or what is more commonly though wrongly called distemper. Probably other diseases are similarly disseminated, and not only diseases but putrefactive organisms, that give rise to open sores that seriously affect the health of stock. One small tick, a species of *Ixodes*, is considered by many observant farmers to be responsible for a form of paralysis in sheep common in several eastern districts; and much lameness in stock, both large and small, is directly due to deep-biting ticks like the Bont Leg and the Bont.

Of all the various ticks probably none is so pernicious in its effects as the Bont tick (*Amblyomma hebraeum*), and it is against this species that most attempts at tick destruction in Cape Colony have been specially directed. This tick is very abundant along the Fish River Rand in the northern part of the Albany district. It was here that the first really systematic and intelligently directed efforts at tick destruction in South Africa, of which I am aware, were begun. The man who instituted this work was the Hon. Arthur Douglass, M.L.A., and the remedy he adopted and still faithfully follows is paraffin oil spraying.

For many years ticks had been getting steadily worse on

Heatherton Towers, Mr. Douglass's property, and along in 1896 he concluded that he must manage to suppress the pest or soon cease cattle-farming. The various dressings that were and are still more or less used by others were tested, and one by one discarded as inefficient. A dipping-vat was con-



FIG. 41.—Spraying horses (Douglass's race).

structed and some dipping tests made, but various circumstances led to the abandonment of dipping in favour of spraying. His efforts to find a successful wash were finally rewarded by his demonstration that paraffin oil, the ordinary illuminating oil found in every household, answered the requirements. Other colonial farmers had used paraffin

against ticks, but when put on with a sponge or cloth, as was usual, it was less effectual against ticks, and burned the skin of the animals. In America, too, paraffin had for several years been employed as a cattle spray, but Mr. Douglass did not know of this, and the fact should not detract from the

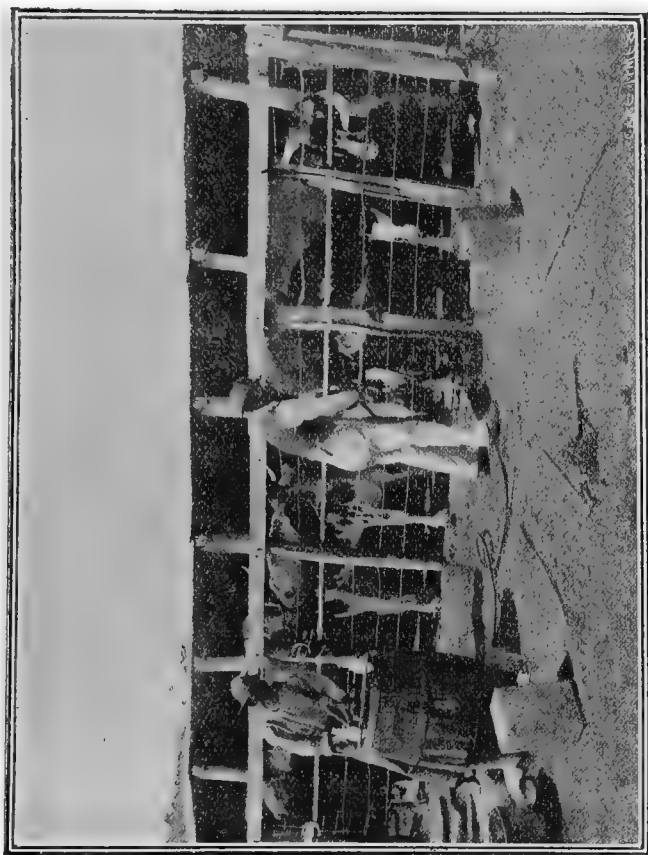


FIG. 42.—Spraying cattle (Douglass's race).

credit given to him. His delight at the discovery was unbounded, and with characteristic enthusiasm he issued an invitation early in 1898 to all and sundry interested to visit his farm on an appointed day to witness a practical exposition of tick-killing by the newly-found agent. The value of the demonstration was not wholly the bringing forward of oil as a

spraying material, for of almost equal importance was the exhibition of how a wash could be economically and quickly applied by having the cattle being treated enclosed in a narrow passage between two fences.

A large number of farmers attended the demonstration, and by them and the newspaper reporters a knowledge of the remedy was quickly disseminated throughout the surrounding country. Many farmers have begun to follow his example, but not nearly the number that should do so or that would have done so had it not been for the troublous times through which the Colony has but just emerged. Now we may expect a more general crusade against ticks, and hence it is a most fitting time for the review of this oil remedy. The first requisite when many cattle are to be sprayed is the passage in which to enclose them. This is called the "race." Two races are shown in our illustrations, the one at Heatherton Towers, and the one at Cottesbrook, Adelaide, Mr. L. J. Roberts's farm.

The Douglass race is a passage 23 feet long and 2 feet wide, between wires. It accommodates three adult cattle at a time. The posts for the sides are set about 6 feet apart and are bedded in cement. They are strung with eight horizontal size No. 6 wires stayed with three ties of doubled No. 8 wire between each two posts. The top wire is 5 feet from the ground. The floor is made 9 inches to 1 foot thick, and is finished with cement mixed with good sand in the proportion of one to three. It is depressed along the middle, and slopes towards one end, where it drains into a cemented hole at the side. To enable the men to better spray the under side of the animals, a trench about 2 feet wide and nearly as deep extends along each side close to the fence; therein the men crouch who handle the spraying nozzles. A heavy swing gate closes the end of the race when cattle are enclosed, and long poles inserted on the wires are placed between the cattle to hold them apart. The high iron fence at one side of the race shown in the illustration, was erected as a breakwind, that heavy winds might not interfere with the work.

The Roberts race was built much later than the Douglass race, and naturally embodies some improvements. In fact, Mr. Roberts first built a temporary race in close accordance



FIG. 43.—Spraying cattle (Roberts's race).

with the Douglass specifications, and erected his present substantial one after he had gained sufficient experience with the first to know just what was wanted. The race is built in his calf-kraal against the front of his long calf-shed, the supporting posts of which are utilised so far as they are in place. In extreme length—that is, from the front of the first post to the back of the last—it measures 21 feet. Three cattle at a time are accommodated. The front beast is allowed 3 inches short of 8 feet in which to stand, and the second and third beasts each  $6\frac{1}{2}$  feet. There are eight posts in all on each side. These are not spaced quite uniformly, but so as to have one close behind each animal as it stands in place, and others intermediate. There is a single intermediate one in the hind and middle sections, and two in the longer front section. The nearness of the posts to one another obviates the necessity for wire lacings. They stand 5 feet high, and are connected by a beam of heavy native wood, about 2 inches by 6, which extends along their tops outside. This beam is bolted in place and forms an efficient brace. Three feet and 3 inches above the floor—that is, mid-flank high on an ox—a gas-pipe of  $1\frac{1}{2}$  inches diameter extends along the sides of the race on the inside of the posts, the posts being cut away slightly to receive it. Between the floor and the gas-pipe are four heavy wires (the lowest is missing in the illustration), and between the gas-pipe and the brace above is another. I believe Mr. Roberts later decided that there should be two above the gas-pipe, but only one was up when I last visited the place. The wires are let through the posts as is usual in South African fencing.

The width of the race between the two lines of gas-pipe is 24 inches, which is just wide enough to take the largest ox on the farm. The front of the race has a Roberts Patent Gas-Pipe Gate about 3 feet wide by 5 feet high. The floor consists of set stones covered with 2 to 3 inches of cement, and, like the Douglass, it is graded to drain down the middle and into a cement-lined excavation at one side. The cover to the hole may be seen in the illustration of the empty race near the bottom on the right. A hard floor is a necessity. A soft floor quickly becomes a quagmire, and even a cement one soon becomes fouled during spraying by the accumulation of dung, urine, and dripping spray.



The animals are kept separate in the race by short poles passed between them through wire loops in front of the posts. The loops can be plainly seen in the illustration showing horses in the race. No poles were inserted on this occasion. The animals naturally press forward, and the back beasts get their heads over the separating poles. The front one is held back by the gate, and hence the reason for making its section of the race longer than the others.

Behind the race proper is the crush-yard, which leads from the kraal. This yard has strong side fences that converge towards the race, as may be seen in the illustrations taken from behind. A dozen or more cattle, as may be convenient, are driven into the crush from the kraal, and then as they are wanted they are urged along into the race. From the front of the race the exit is into a camp.

Horses and mules are passed through the race, and are sprayed just as are cattle by both Douglass and Roberts. The latter keeps no sheep, and the former only 200 or 300, which are hand-dressed. The ostriches at Heatherton Towers are, however, sprayed with oil and water for the ostrich fly (*Hippobosca struthionis*) and lice, but not for ticks, of which the birds are able to keep themselves fairly free.

The first time animals are driven into a race they are apt to go very unwillingly, and when once in may make violent exertions to jump over the front or sides, or to crawl out beneath. A heavy hard-wood gate on the Cottesbrook race was quickly smashed by a vicious ox, but though the gas-pipe one has been charged and jumped on times without number, it has resisted every strain without being injured. The race gate should be kept open when the animals are driven in until the foremost is in his place, and then it should be quickly shut in his face, and a pole put behind him before he can retreat. After having been sprayed a few times, however, there is little trouble in urging the animals on, the wildest of them generally moving quietly forward in their turn as if conscious of the benefits of the treatment.

Some farmers do not use a race in spraying, but tether their cattle to posts in the kraal instead. When only a few tame cattle are concerned, this procedure is not a bad one, but it is cruel and exceedingly laborious when a large lot of half-

wild cattle have to be handled. Milch cows that are stabled (as few are outside the towns at the Cape) may be sprayed in their stalls, but they should be turned out immediately afterwards.

The time required to spray cattle in a race after the men and animals have become accustomed to the routine averages about a minute a head. Sixty animals an hour are ordinarily handled at both Heatherton Towers and Cottesbrook with sprayers working simultaneously on the two sides of the race. Seventy-five to 100 animals can be hustled through, but the first requisite of cattle-spraying is thoroughness, and to maintain thoroughness the men at the nozzles must not be unduly rushed.

Five or six men are needed for the work, one or two at the pump or pumps, one on each side to spray, one behind to drive, and one to attend the forward gate. The most trustworthy men should be at the nozzles, for the ticks that are not hit by the spray will continue to live. The animals are not sprayed all over.

Only the under and rear parts, the dewlap, tail, feet and legs are generally sprayed, for few adult Bont ticks are found elsewhere. The men keep a sharp look-out, however, and wet down any that chance to be high on the flanks, and may spray the whole body if the blue tick is abundant. Bont ticks on the sides and back, and along the tail, are more common amongst sprayed cattle than unsprayed. This is because males that get in these unusual positions may be overlooked in spraying, and the other males being killed, the females search farther in quest of mates, thus finding those in the exceptional places which ordinarily are quite overlooked.

Mixed oil and water soon superseded the use of pure oil, and for the application of the two liquids together specially fitted pumps are necessary. Mr. Douglass was using pure oil when the photographs here given of his race were taken, and hence the pump shown is a simple one. At that time his race had an extension to hold three cattle after spraying, that the oil dripping from them might be saved; but with the use of diluted oil it became of small advantage to save the extra drip, and the extension was removed. Mr. Douglass uses two

Douglass Patent Syphon Pumps for his spraying, whilst Mr. Roberts has one large pump that he fitted up himself; it delivers two streams, one of which is led through an iron pipe laid under the race for the supply of the hose on the farther side.

Mr. Douglass has his pumps set to supply 25 per cent. of oil. Mr. Roberts sets his at 20 per cent. Experiments which Mr. Roberts and the writer conducted showed that 15 per cent. mixture well applied was fatal to the ticks, but no pump always works perfectly true to its gauge, and hence some allowance is necessary.

The quantity of oil used at Heatherton Towers approximates 10 gallons to 100 head of mixed cattle, and at Cottesbrook the average is a trifle higher, being about  $10\frac{2}{3}$  gallons. Ten gallons in a 20 per cent. mixture for 100 cattle averages  $\frac{1}{2}$  gallon of liquid to a beast. The amount of oil recovered from the drain-holes is usually from 5 to 10 per cent. of the total used. Mr. Douglass has all the drainings poured into a large tank and has all the oil skimmed off after twenty-four hours. Mr. Roberts generally boils his before skimming, and thinks he saves by the method.

The interval between sprayings is made fourteen days at Heatherton Towers and twelve days at Cottesbrook. Mr. Douglass at first thought one spraying a month would do, but soon found the interval too long, and reduced it to nineteen days. The idea is to spray sufficiently often to prevent any female Bont ticks from maturing. In 1899 the writer traced the life-cycle of the Bont tick, and found that the female took six and a half days or longer to fill up on an ox. Few were found to fall until eight days had passed. It so happens, fortunately, that a female rarely attaches except by a male that has been on several days, and that when one does attach elsewhere its development is very slow until after it has contrived to mate. No observed males have become attractive until they have been on four days, and few have attracted until they have been on five or six. Therefore the period after a thorough spraying, during which no female can mature, may be calculated as twelve days, which allows for a male attaching the day of spraying and of becoming ready for company on the fifth day, and for a female then at once

finding this male and becoming ready to drop on the last day. It seems unlikely that any females will actually fill up in the minimum time, particularly as one would imagine the presence of the oil to have a deterrent effect for a day or more, and it was thought quite safe to make the interval between sprayings an even fortnight. Mr. Douglass adopted this interval soon after the tick studies were published, and seems still satisfied that it is short enough. Mr. Roberts, however, after two years' experience, fears a few females are permitted to mature, and has now cut off the two extra days. It must be remembered that if any Bont males escape a spraying, new females may mate with them at once, and perhaps be matured in seven or eight days; the necessity for very thorough spraying is thereby emphasised.

The common blue tick stays on an animal three weeks or more; very few specimens indeed come off under twenty-three days. Hence the complete destruction of all that come on the animals once in three weeks should prevent any of this species from fully maturing. The females of the red tick, the species found clustered about the anus and genitals, may go on coincidently with males, and drop off six days later fully engorged, and hence dressings for it would have to be at least every six days to be most effectual; but, fortunately, this species is not one that requires repressive measures. It spends a fortnight or more in the ears of animals in its early stages, and could there be destroyed at longer intervals were it not that syringing ears with paraffin is too harmful a measure to warrant adoption.

The effect of oil-spray on the ticks begins to be apparent almost at once, and in an hour after spraying most of the victims are dead. On the next day many are already dry, and easily break off. Engorged female blues become shrivelled, blackened, and hard, and look as if they had been parched. It is not easy to determine the full effect until after twenty-four hours, so judgment on the success of a spraying should be suspended for that length of time.

The Bont tick is as quickly destroyed by the spraying as any other kind. The red tick is the most likely to escape, as the skin where it prefers to attach is devoid of hair, and the spray therefore fails to remain. The blue tick, when actively

feeding, is almost certain to die; but when it is hanging dormant, waiting for a change of skin, it stands a good chance of escaping. The blue is the only species of tick that we have that always remains on the animal at such times. The efficacy of the spray largely depends on its being held on the animal in contact with the ticks for a short space of time. It is the oil on the hair, rather than that on the skin, that does the killing. Oil rubbed on with a cloth is apt to burn the skin, and yet not kill all the ticks, for the hairs get little of it. But an oil-spray should be fairly hard—hard enough to drive against the skin—or the bases of the hairs will not be wetted, and many young ticks close against the skin will escape. The Bont tick will often survive a momentary immersion in pure paraffin, and this anomaly has made some people doubt the efficacy of spraying; but they have only to try spraying once to discover how differently the spray acts to the momentary dipping.

The effects of oil-spraying on the animals is generally not injurious in any respect. In hot, dry weather the oil soon evaporates, without causing any damage to the skin, even when sprayed on undiluted. When cool, cloudy weather prevails, or when the spraying is done late in the day, a slight inflammation of sensitive skin may result, however much or little oil is used; but this injury is rarely enough to cause any suffering. Those who spray naturally take reasonable precautions to avoid injuring the animals. As far as can be arranged, the spraying is done early on fine days, and stabled animals are allowed to dry before they are again put under cover.

The oil-water mixture seems far less apt to inflame the skin than paraffin emulsion—that is, the milky mixture formed by churning paraffin in strong soap and water—and the simple mixture is more destructive to the ticks.

As ticks have decreased on their farms, both Mr. Douglass and Mr. Roberts have ceased to spray in the winter. Mr. A. W. Douglass, son of the Hon. Arthur Douglass, who is now farming Heatherton Towers, sprays during eight months only—September to April inclusive. He says very few ticks are now seen on the cattle during the winter. Mr. Roberts has his cattle brought up to the race at the regular periods,

and simply hand-smears the few ticks found on them. Three years ago the Bont tick was numerous on his cattle all through the winter, and many of them were studded all over with the blue species even in July.

How long must spraying be continued? No strict time-limit can be put. The chances are that the remedy as practised by Mr. Douglass will be needed, without any relaxation, at least through the summer months, for all of three years, and perhaps five. After one season of faithful spraying a decided improvement will be evident; but even after five years it is probable that several sprayings every season will be necessary, and necessary indefinitely thereafter, to keep the pest well suppressed.

Ticks have now assumed such importance in veterinary pathology that it would be impossible to give a description of them here, and the reader is referred to books devoted to this subject.

*Conclusions.*—(1.) Texas cattle fever is a disease of the blood, characterised by a destruction of red corpuscles. The symptoms are partly due to the anæmia produced, partly to the large amount of *débris* in the blood, which is excreted with difficulty, and which causes derangement of the organs occupied with its removal.

(2.) The destruction of the red corpuscles is due to a micro-organism or micro-parasite which lives within them. It belongs to the protozoa, and passes through several distinct phases in the blood.

(3.) Cattle from the permanently infected territory, though otherwise healthy, carry the micro-parasite of Texas fever in their blood.

(4.) Texas fever may be produced in susceptible cattle by the direct inoculation of blood containing the micro-parasite.

(5.) Texas fever in nature is transmitted from cattle which come from the permanently infected territory to cattle outside of this territory by the cattle tick (*Boophilus bovis*).

(6.) The infection is carried by the progeny of the ticks which mature on infected cattle, and is inoculated by them directly into the blood of susceptible cattle.

(7.) Sick natives may be a source of infection (when ticks are present).

(8.) Texas fever is more fatal to adult than to young cattle.

(9.) Two mild attacks or one severe may prevent a subsequent fatal attack.

(10.) Sheep, rabbits, guinea-pigs, and pigeons are insusceptible to direct inoculation. (Other animals have not been tested.)

(11.) In the diagnosis of Texas fever in the living animal the blood should always be examined microscopically, if possible.

The regulations of the American Department of Agriculture provide for the prevention of the introduction of the disease into non-infected territories by the complete isolation of all cattle from infected territories between March 1st and December 1st of each year ; on the proper disinfection of the litter and manure from such cattle during transportation. Furthermore, such cattle can only be transported into uninfected territories for immediate slaughter during the prescribed periods.

There are further directions in the American Report, from which the foregoing has been compiled, without the scope of this work. The author may, however, state that the Report, covering 126 pages of closely printed matter, in addition to nine pages of illustrations, is a most valuable one, conducted with great care by Theobald Smith, Ph.B., M.D., and F. L. Kilborne, B.Agr., B.V.S., under the direction of Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, Washington, 1893.

#### IXODIC ANÆMIA IN JAMAICA.

In July last, 1896, I was commissioned to go to Jamaica to study a fatal disease which had prevailed amongst the cattle of that island for the last few years ; and, after careful consideration and two months' experience, during which period I examined diseased cattle in all parts of the island, I arrived at the conclusion that I had to deal with Texas fever, of which the following is a short description.

Cattle enfeebled from any cause seem to form a more favourable habitat to the ticks, and are more predisposed to the disease than the healthy and strong ; but the healthy and strong have no immunity, as they are frequently attacked, and succumb. I have therefore arrived at the conclusion, after carefully weighing all the facts and circumstances which have been brought before me, both directly and indirectly, that the

disease is due to the attacks of ticks, and (with the exception of the Mysore cattle) that no cattle have immunity. Some are but slightly invaded by the ticks; some, owing to strength of constitution or other causes, seem to throw off the effects with but little appreciable suffering or damage, whilst in others the attack is acute, and terminates fatally in a few days after the first manifestation of the disease; and I re-assert that, after carefully weighing the evidence, I have arrived at the conclusion that the disease is, as presented to me, a modified form of Texas fever, now prevailing in other parts of the world besides Jamaica, and that it is carried from place to place by ticks.

The disease in itself cannot be called contagious or infectious; there is, therefore, no necessity for burning the carcasses of dead animals.

Animals suffering from the disease, if *perfectly freed from ticks*, do not transmit the disease, no matter how severe the attack may be; but if invaded by the parasite, the pastures on which they graze are first contaminated by the mature ticks, which drop from cattle, and in about seven days lay their eggs in the grass; the eggs hatch in about other twenty days—sometimes a longer period—and the young ticks are at once ready to crawl on to the cattle. They then become again encapsuled and emerge from the second shell in fourteen or fifteen days.—(See p. 423). If these figures be added together, it will be found that the shortest possible time, after tick-infected cattle are turned out into a field, in which the disease may appear is about forty days; but the period of first attacks may be much longer than this, as all the eggs are not laid upon the same day: there is therefore a daily hatching for consecutive days. (Own observations: Texas ticks placed in bottle on 5th September commenced to lay on 17th.)

It is rather unfortunate that during the earlier part of our visit the animals examined, both prior to and after death, were suffering from the disease in its chronic form, and in which many of the marked symptoms and *post mortem* conditions of acute Texas fever were absent; but during the latter portion of the visit more decidedly marked signs of that disease were observed.

One characteristic sign of acute Texas fever (as described by the American writers)—red water or blood-coloured urine, hæmoglobinuria—was absent. They state that “the one sign regarded



as peculiar and pathognomonic (characteristic) in this disease is the discharge of urine having the colour of blood. This colour is not due to a discharge of blood from the kidneys and subsequent breaking up of the red corpuscles, but to a filtration of the colouring matter of broken-down red corpuscles (hæmoglobin) already in solution in the circulation, into the urine in the excretory structures of the kidneys." This statement is perhaps too obscure to be easily understood. It means that the red blood-cells are broken up in the blood-vessels, and that the colouring matter thus set free tinges the fluid portion (which is naturally colourless), and which, when excreted by the kidneys, presents a red appearance. Now this sign, considered so important by the American authors, and stated by them to be present in 33 out of 46 cases, did not present itself except in one doubtful case. The animal (a cow) was suffering from a very prolonged but not severe attack, and had latterly passed red water. I was of opinion, however, that this tinge was due to some injury to or breaking down of the urinary apparatus, as the colour was due to actual (coagulating) blood, and not to the colour of broken-down blood-cells. The information obtained from all who were acquainted with the disease, that red water is not a symptom, would have caused me much surprise had I not known that in Australia red water is only observed after the animal has been travelled. (One of the constituents of the blood absent in healthy urine, viz., albumen, was found in all the specimens of urine examined.) Other signs of Texas fever, such as enlargement of the spleen and of the liver, sero-sanguinous or red watery condition of the fat, &c. about the kidneys, were absent. The spleen had in no instance shown the enlargement described by the American writers, but the liver in some three cases was slightly enlarged, and presented a pale yellow mottled colour, due to bile congestion and fatty degeneration. When cut into, the tissue presented a mottled appearance throughout, and was bloodless, whilst the larger blood-vessels discharged a very dark-coloured and thick blood. But whilst this characteristic of acute Texas fever was present in these few cases, the liver itself did not present the three to five pounds enlargement over that of health, as stated to be the case in that disease; in fact, we did not find one liver to be appreciably increased in weight, but the

bile was generally increased in quantity and mixed with mucus. The appearance of the heart also differed from that described in Texas fever, the petechial spots passing along the intra-ventricular groove and near the base, extravasation, &c. being absent, but the heart itself was found to be pale and undergoing a retrograde change, as seen in the Texas disease after the subsidence of the fever.

*Temperature.*—In most cases there was more or less elevation of temperature. In one case it was as low as  $101.4^{\circ}$  F.; this was in a very emaciated and debilitated calf. As a rule, however, the temperature ranged from  $103.5^{\circ}$  to  $106.8^{\circ}$  F.

*The appearance of the Blood and Tissues.*—Animals that were grazed upon morasses and boggy grounds were upon *post mortem* examination found to have flukes (*Distoma*) in the liver, and in some cases in the lungs, but, except in one case, the liver flukes were few in numbers.

In districts in which there are no rivers, and where the drinking water is obtained from ponds supplied by the rains, the *Strongylus contortus* was found in varying numbers; but we concluded that, though they might aggravate the conditions, they were not the cause of the disease, as in the majority of the *post mortem* examinations neither flukes nor strongyles were present. In two cases the lesions of broncho-pneumonia—as seen in American and Canadian cattle—were found, and these were supposed to be tubercular. No tuberculosis was met with during the whole investigation.

Where the cattle obtained water from wells and running streams, the *post mortem* appearances were as follows:—The organs and tissues did not to the naked eye materially deviate from those seen in health. There was bloodlessness (anæmia), characterised by pallor of the organs and some degree of degradation of their structures. The blood, however, was thin, watery, pale—in some instances scarcely tingeing the fingers of the observer—whilst microscopically it presented those characteristics reported upon by my son, Dr. Williams.

The symptoms during life were—1st. great depression, the animal presenting signs of languor and debility, the appetite was indifferent, and rumination irregular and infrequent; there was emaciation, the loss of flesh being often extreme; the ears drooping, and the visible mucous membranes, *i.e.*, those of the

nose, mouth, eyes, &c., exceedingly pale. In one uncomplicated case only did we observe yellowness of these membranes. There was sometimes diarrhoea, but we did not witness one case in which there was constipation. The pulse in all cases was exceedingly small and weak, ranging from 65 to 120 beats per minute. About 85 beats, however, or about twice the natural number, seemed to be a medium. The urine was pale in colour generally, and in all instances when tested was found to contain a small amount of albumen. The breathing, except in calves, or when the animal was disturbed, was about 20 movements per minute, or about one-third more than the natural number. There was no cough nor a discharge from the nose, or other signs of disease of the lungs, except in the cases already cited. The eyes had a sunken appearance, and the ears drooping, and the animals had a staggering gait, particularly if hurried or excited. In several instances they fell to the ground when moved sharply or turned round suddenly; in one instance the animal fell down dead, so great was the general prostration and weakness of the heart's action.

The skin about the shoulders, lower portion of the neck, and other parts easily rubbed by the animal, was generally denuded of hair, and in many instances presented a rough scaly appearance. This symptom has been described as one preceding the advent of the tick, but such is not the case, as it was found upon close examination that the skin was covered with ticks underneath the undisturbed hair of the host. I therefore conclude that this symptom is a sequence to and not a precursor of the disease, as supposed. Some penkeepers call this "mange," and seem to think that it has no connection with the disease. On close examination it was found that there were not only fully matured parasites present, but many in an embryonic stage,—that is to say, covered by a shell of thin membrane; upon breaking which, two young ticks, namely, a male and a female, were usually found in close contact, the male being of a brownish colour and having four pair of longish legs, the female larger, also having four pair of whitish legs, and paler in colour than the male.

The surface of the body was generally more or less covered with ticks, which presented at least three varieties, viz.—1st. the large blue cow tick, also called the dog tick—*Ixodes ricinus*—the

first tick known on this island, and not supposed to be injurious ; 2d. the silver shield tick—*Ixodes scapulatus*—which clings much more tenaciously to its host, and is difficult to remove. These two kinds of ticks, as well as the grass lice, or the young ticks of both kinds, are supposed to be non-injurious to cattle, but I cannot subscribe to this, as they must do harm, and at least assist in the induction of the anæmia or bloodlessness which follows. The third form—the one stated to have been unknown by the majority of penkeepers and others until pointed out to them—was brought before my notice about the third day of our investigation, having been found shortly before upon a lot of cattle badly attacked by the wasting disease. Upon examining this tick I arrived at the conclusion that it was very similar to, or identical with, specimens of the Texas cattle tick.

During my investigations into a tick disease in Scotland, it was found that old and withered grasses and decaying vegetable matter of all kinds formed a cover for the ticks, and that they preferred to deposit their eggs where there was an abundant cover, and thus secure their non-destruction during the cold of winter. Although protection against low temperature is not necessary in Jamaica, there is plenty of evidence to show that when decaying and withered vegetation is allowed to remain on the ground there the ticks will be found, and I have been informed, over and over again, that during some parts of the year cattle will not face guinea-grass ; that when placed in such pastures they will show signs of excitement, jump over the fences, and thus abandon even the most luxuriant pasture. Now this proves that the ticks are there in abundance, and no wonder, when it is considered that guinea-grass, said to be eaten down, leaves a withered stubble often two or three feet long, which forms a cover for the tick to deposit its ova upon, where they hatch, and are ready for the first cattle they may come in contact with.

I therefore think that when cattle are removed from guinea-grass the stubble should be destroyed by burning.

I am told that, when guinea-grass is burnt standing upon the ground, the destruction is imperfect, and that the heat simply favours the hatching of the young ticks, the ova or eggs being under the stones, &c. If this be the case, the stubble should be

cut down, collected into heaps, and burnt, the ashes remaining being spread over the land, and thus return something back into the soil. If it be proved by analysis that the soil is deficient in some essential ingredient, this should now be applied, as fertility of the ground not only insures better crops, but renders that ground unfavourable to ticks and other parasites, as proved in Scotland by liming and otherwise improving tick-infested pastures. In addition to the destruction of all guinea-grass stubble, decaying and dead vegetations of all description should, when possible, be burnt, and the ashes spread over the land. During my travels in Jamaica I have witnessed many thousand tons of all kinds of vegetable matter allowed to remain on the ground, harbouring vermin and preventing the growth of useful materials; whereas, by collecting such refuse into heaps and burning, not only would the harbour for vermin be destroyed, but the ashes so obtained would to some extent, by improving the land, compensate for the trouble and outlay.

*With regard to the destruction of the Ticks themselves.*—A united effort must be made to remove the cause of the disease, by diminishing, or, if possible, wholly destroying the ticks; and for the purpose of killing them on their host I have, after due consideration as to the cost, and knowing full well that the profit on an £8 to £10 four-year bullock will not allow much expenditure of money, to recommend the following treatment; but, if money be not spent and an earnest and united effort be not made, I cannot speak as to the future consequences.

For the destruction of the ticks on the cattle I recommend the following as the cheapest and most reliable dressing:—to every 4 gallons of linseed oil add 1 gallon of tar and 1 lb. of resin. The tar and resin to be melted with a little of the oil before being added to the bulk, and applied to all parts of the tick-infested skin; if one dressing be not sufficient, a second should be applied in a few days. All large ticks might be removed by being picked off and carefully destroyed, not thrown upon the ground and stamped upon, but burnt or smothered in tar. Oil destroys the tick by suffocating it, and any oil will do this, but boiled linseed oil is a cheap oil, and being a “drying oil,” will remain longer on the surface of the body than a non-drying oil. Jeyes’ Fluid and Sheep Wash have proved of service in destroying the ticks, but the mixture of linseed oil, &c. will

answer the same purpose and be more permanent in its effects.

In America and Australia cattle-dipping is now resorted to, and where the cost can be afforded it should be done in Jamaica, for after all it must be confessed that to see the cattle attendants dress any animal is but a sorry sight. If the dressing is not thorough, it is money thrown away; every crevice fold, and depression should be thoroughly dressed, the cavities of the mouth, &c. examined, the ticks removed, and the parts dressed; the eyelids should also be carefully examined, and the ticks gently pulled off.

Every effort should be made to conserve and increase "tick-destroyers," such as the Black Tick Birds, of which I saw two kinds, one a long-billed and the other a short-billed bird. I look upon these birds as the greatest friends to the cattle owners. I have had much amusement in watching these birds, as there seems to be an understanding between them and the cattle, whereby they are assisted and encouraged to destroy the ticks.

The domestic fowl is also very valuable as a tick destroyer, and for this purpose it could economically be kept in increased numbers. The long-legged varieties have a great advantage over the shorter legged ones, as they can reach much higher and thus remove more ticks, which usually do not favour, at least to any great extent, the lower parts of the limbs. Tick-destroying birds, such as the starling and perhaps the song-thrush, might be imported.

To a stranger visiting the island the scarcity of birds is a striking feature. I have been told that it is due to the mongoose, which has not only diminished the number of wild birds and domestic fowls, but other tick-destroying creatures, such as the ground lizard. Now this destruction of the natural tick-destroyers should, as far as possible, be prevented,—1st, by legal protection, 2d, by encouraging the slaughter of the mongoose, a small reward being offered for every head brought to officers appointed for the purpose of receiving them.

For further information see my Report to His Excellency the Governor of Jamaica, *Veterinary Journal*, December 1896.

## TEXAS FEVER (IXODIC ANÆMIA).

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*Photo-Micrographs of the Hæmatozoon in various stages.*

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The subjects of the following Photo-Micrographs have been worked out and photographed by my colleague, Dr. Hunter, from specimens prepared in Jamaica by my son Thomas A. Williams, M.B., C.M.

All these illustrations are from 1500 to 1800 diameters, except 51, 54, and 55, which are 2100 diameters.

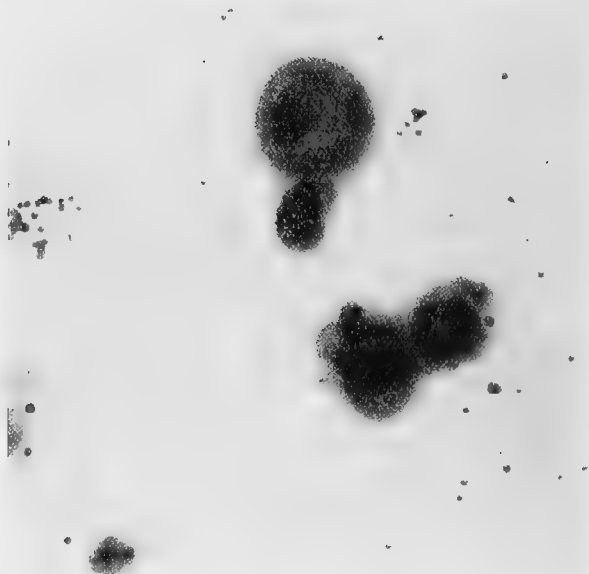


FIG. 44: STAGES 1 AND 2.—Blood of Blue or Cow Tick, showing blood corpuscles and *plasmodia* of various sizes, chiefly in the plasma; average size about  $1\ \mu$  in diameter; the smaller bodies are the more highly refractive.

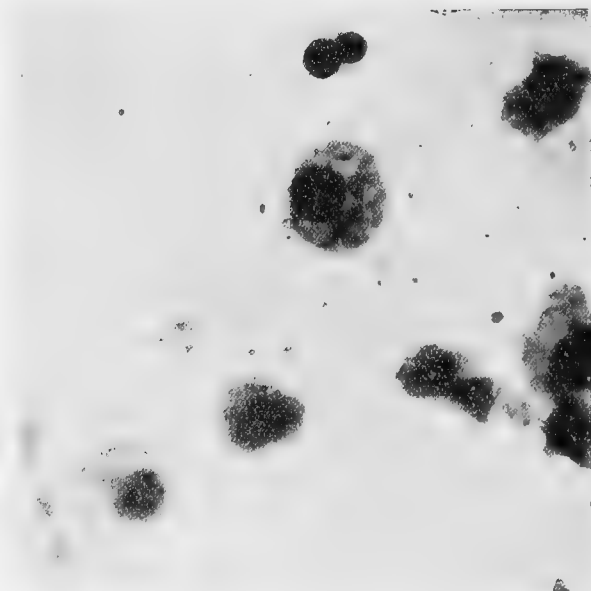


FIG. 45: STAGE 3.—Blood of Silver-Shield Tick.—Small segmenting bodies (*plasmodia*) in interior of the blood corpuscles. This may be called the third stage of development.



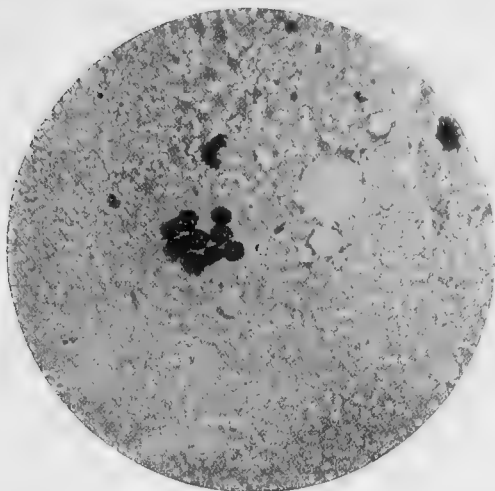


FIG. 46: STAGES 1 AND 2.—Blood of Texas Tick, showing plasma crowded with small highly refractive bodies; average size  $3\mu$ . These highly refractive bodies appear to be the most easily communicated form of the organism. It will be observed that these "coccus-like" bodies are much more numerous in this than in the two previous illustrations (Figs. 44 and 45); a cluster of spherical segmenting bodies, considerably pigmented, and coccidioidal in appearance, is seen near the centre; the blood corpuscles appear to have undergone but slight changes.

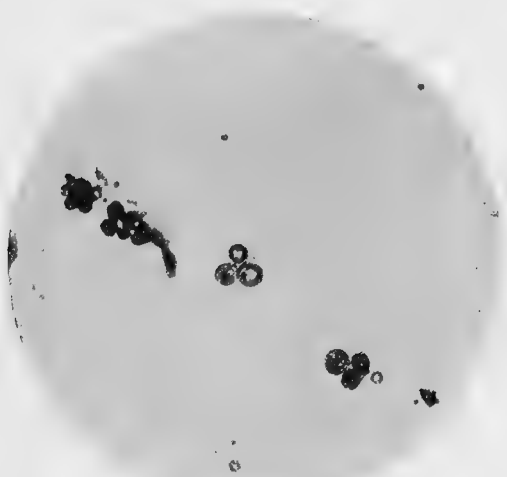


FIG. 47: STAGES 1, 2, AND 4.—Blood of Texas Tick.—A row of the segmenting bodies in various stages (the small round bodies being thrown out of focus). The segmenting pigmented bodies seen in this and 46 may be called the third stage, although there is much difficulty in following the developmental changes.

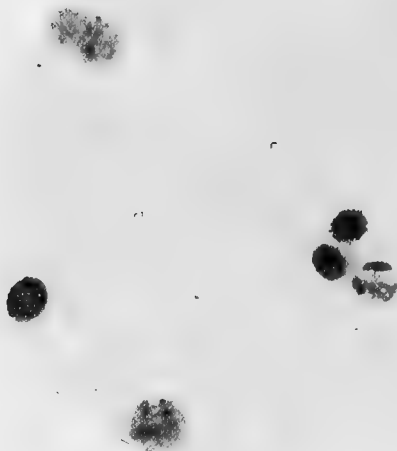


FIG. 48: STAGE 3, SAME AS SEEN IN TICK FIG. 45.—Blood of Ox, showing small segmenting bodies(plasmodia) in interior of the red blood corpuscles. These stain with great difficulty, and are here represented by the colourless (white) portions. The red corpuscles are shrunk to about half their normal size, the shrinking being more pronounced than in the corpuscles of the "Tick."

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FIG. 49: STAGE 4.—Blood of Ox, showing enlargement of free segmenting bodies, the previous stages of which are seen in Figs. 46 and 47. The bodies are seen here perfectly spherical, with distinct and smooth outlines, and show, apparently, no internal structure. They are not, however, vacuous, but contain transparent endoplasm, which is less refractive than the small spheres noticed in the first four illustrations. They vary in size from  $1\ \mu$  to  $1\frac{1}{2}\ \mu$  in diameter.



FIG. 50: STAGE 5.—Blood of Ox.—A group of segmenting bodies in a stage succeeding that of Fig. 49. Here traces of internal segmentation are visible, and the outlines are slightly notched.



FIG. 51: STAGE 6.—Blood of Ox.—A more advanced stage of segmentation, where the organism is distinctly breaking up into pear-shaped segments—the so-called "Marguerite" stage—the usual number of "leaves" being apparently from five to eight.

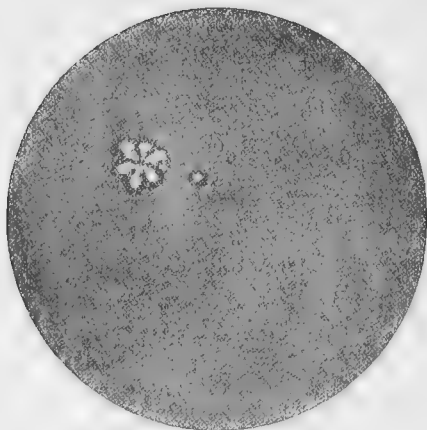


FIG. 52: STAGE 6.—Blood of Ox.—From seat of fresh bite of Texas Tick, with "dark background illumination," showing a very perfect "Marguerite" of six leaves, and a small refractive sphere. The blood cells are faintly seen, with contained plasmodia.

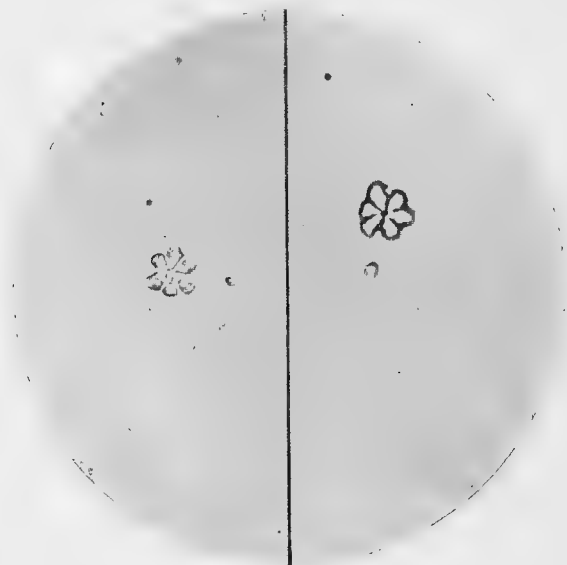


FIG. 53: STAGE 6.—Two other views of Fig. 52 under different illumination and focus; the altered blood corpuscles being better seen than in 52.

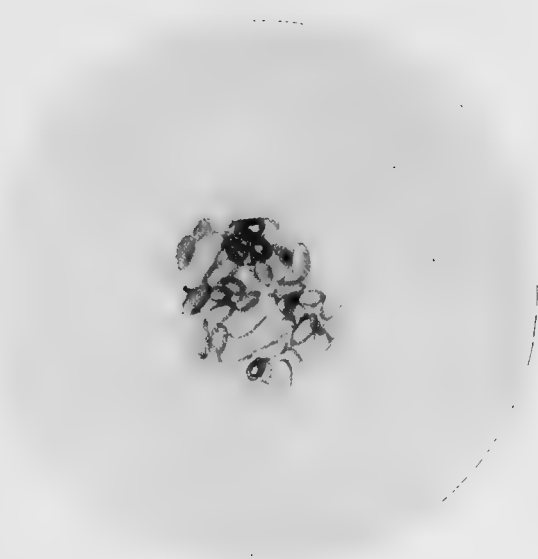


FIG. 54: STAGES 7 AND 8.—Blood of Ox, showing at least two stages with intermediate transitions, the seventh stage being simply the enlarged pear-shaped bodies of Figs. 51, 52, and 53, which are here becoming ovoid and lengthened; the last here seen, or eighth stage, being banana-like bodies almost straight, very pellucid, with condensed protoplasmic extremities. It is believed that these condensed ends give origin to the flagella, as is the case in similar Protozoa; the intermediate forms show various stages between the seventh and eighth; some of these show large vacuoles, the meaning of which is at present uncertain. This stage stained with difficulty, and the contents seem mostly fluid, their average width being a little over  $1\mu$ .

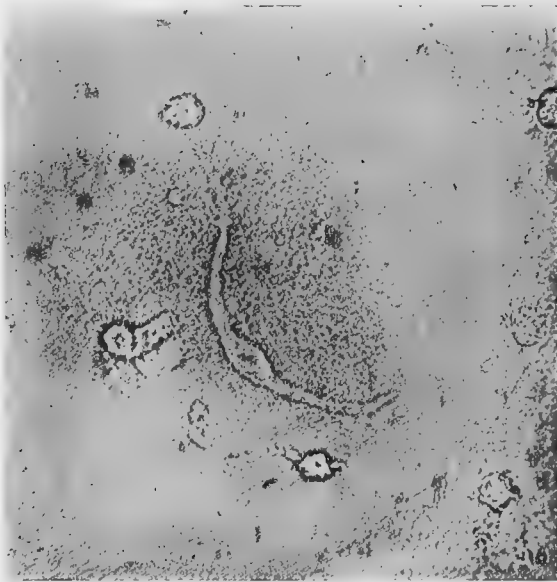


FIG. 55: STAGE 9.—Blood of Ox, showing what resembles the mature stage of the Protozoon *Pyosoma bigeminum*. This flagellate stage must be rarely reached in cattle, for there are very few seen in the specimens; but they may probably be more numerous in acute cases, none of which were seen by me. The plate shows various stages of the disintegration of the monad, which seems extremely liable to break up after removal from the body. (Indeed this flagellate stage is believed by some to be one of degeneration.) The American observers seem evidently to have failed in successfully studying this stage, except in perfectly fresh blood, and recommend observations upon it to be made in the field. That in the lowermost position in the plate shows a mere trace of the shrunken flagella with considerable alteration and shrivelling of the central portion; the others in the plate, particularly that on the right hand, appear to be still further degradations of the mature form; the central dark spots in the figures are purely optical, and do not represent nuclei. The flagella, seen in the central figure, being long in proportion to the microscopic power used, cannot be represented in focus throughout the whole length in a photograph, as they are not all on one plane. It will be difficult to establish the identity of this stage without further extensive observations on Texas Fever, as well as upon the disease Surra, seen in the horse in India. (See *Surra*.)

*Summary.*

(a.) That the disease as witnessed by me is a chronic form of Texas fever conveyed from place to place, and transmitted from one animal to another through the intervention of the tick.

(b.) The infection is conveyed by the progeny of ticks which have matured on infected cattle, and is inoculated by them directly into the blood of susceptible cattle.

(c.) It is stated that two mild attacks, or one severe one, will probably prevent a subsequent fatal attack.

(d.) That animals under one year old, though not immune, are not so frequently attacked by the slower form of the disease, and that if such be nursed over one or more mild attacks, they may probably obtain immunity. Ticks being the cause of the disease, there should—

- (1.) Be a united effort to destroy them on the cattle by external dressings, and on the ground and pastures by burning.

“Dipping ponds,” as now used in America and Australia, should, where possible, be preferred to the applications by hand. Plans of the most recent and most effective ponds or baths should be obtained.

- (2.) To prevent the further introduction of ticks into the country by an effective system of quarantine.
- (3.) After destroying the ticks on the cattle, the “animal strength” should be maintained by keeping them in a quiet sheltered place and feeding well, without causing them to travel for their food.
- (4.) To avoid drastic medicines. If the cattle drink pond water, it will be safe to conclude that the disease may be complicated and aggravated by the *Strongylus contortus* in the fourth stomach, for the destruction of which a dose of sulphate of copper should be administered, and, if necessary, repeated two or three times; but when cattle obtain their water out of running streams, or from clean tanks, the copper is not necessary. If the animal be feeble, the following may be administered to act as a tonic and as a food, viz.:—Sulphate of iron, nitrate of potash, and common salt (chloride of sodium), of each one drachm in a pint of pimento tea, once a day for a week or so.

## EQUINE PIROPLASMOSIS, OR TROPICAL BILIOUS FEVER, IN INDIA.

It is not yet known how the piroplasmata are introduced into the system, whether by ticks (which is the most probable) or by other agents, such as flies.

The disease is characterised by high fever—temperature  $105^{\circ}$  to  $106^{\circ}$  F.—great depression, and hurried respirations; the animal hangs his head, eyes closed, and a flow of tears runs down the face; the visible mucous membranes are a pale yellow colour. The patient is usually constipated, and the urine is of a deep amber colour, or even a brownish-black, and sometimes even sanguineous.

The mucous membranes become of a dirty yellow colour, and that of the membrana nictitans has upon it numerous ecchymoses of a purplish hue; these increase in area, and may finally coalesce in the membrana nictitans and become quite purple, though the conjunctiva of the lids remains a dirty yellow.

At about this period—namely, five days after the commencement of the attack—there appears some cedema of the limbs.

About the sixth day the purple colouring of the membrana nictitans gradually disappears, leaving the membrana of a dirty yellow colour. During this period the temperature varies, rising in the evenings up to  $103^{\circ}$  F., and falling in the morning to  $100^{\circ}$  F.

Urticaria is not an uncommon accompaniment, as indicated by such swellings of large size along the lower parts of the chest and abdomen and of the sheath, and sometimes smaller eruptions in the limbs, which may persist for weeks. A most noticeable feature is the capriciousness of the appetite, and the progressive wasting of the muscular tissues, accompanied by great general debility (Captain H. J. Axe, A.V.C.).

Sometimes this disease occurs in a chronic form, and animals are subject to repeated attacks of jaundice and hæmo-albuminuria.

MALIGNANT JAUNDICE OF THE DOG<sup>1</sup> (TICK-FEVER).

This disease is known under several names by veterinarians, as, for instance, bilious fever and malignant malaria, but the term here adopted, malignant jaundice, is the one preferred

<sup>1</sup> Abstract from a report by Mr. Chas. P. Lounsbury, Government Entomologist, Cape Colony, 1901.



by Dr. Hutcheon, the Colonial Veterinary Surgeon. Cape Dutch apply to it the vague name "hondziekte"; while colonials of British birth often speak of it as "distemper," under the mistaken impression that it is identical with a canine malady of that name that occurs in England. Dr. Hutcheon records in his annual report for 1899 that malignant jaundice was first brought to his attention in 1885 at Port Elizabeth, and that it has since reached Cape Town, and spread to various parts of the Colony and adjacent territories along the main lines of traffic. It now ranks as the most fatal form of disease affecting dogs in the Colony. The animal attacked first shows its illness by a rise of temperature. It loses appetite, becomes listless, and lies quietly in one position for hours at a time; soon it refuses to eat at all, but may drink much water. The gums and mucous membrane of the mouth become pale and bloodless, and the breath takes on an unpleasant odour. The thin skin of the abdomen often becomes yellowish, the discoloration being due to jaundice of the fatty tissue beneath. If the blood be superficially examined, it is seen to be thin and watery. At a late stage the urine becomes dark red or brownish; this is a most unfavourable symptom, and an animal which shows it has little chance for recovery. Some dogs die within twenty-four hours of indicating illness by their appearance, but most generally linger on for five days or more, and seem to die from sheer exhaustion.

*Disease Similar to Red Water.*—The similarity of the disease to red water in cattle was noticed by Dr. Hutcheon from the first; and hence he was not surprised when, about two years ago, he submitted diseased blood to the Colonial Bacteriological Institute for examination, to learn that the red corpuscles enclosed organisms very similar to those found with the cattle malady. At once the suggestion occurred to him that as red water was spread by ticks, malignant jaundice was probably spread in the same manner.

*Habits of the Dog Tick.*—Ticks are not uncommon parasites of the dog, but all that I have taken from this animal in the western part of the Colony have been of a single species, *Hæmaphysalis Leachi*, Audouin (determined for me by Professor G. Neumann, Toulouse, France). Consequently, sus-

picion at once attached to this species as the possible agent for transmitting the disease. This dog tick differs widely from the cattle tick in some of its habits, and one essential difference is in regard to its place of moulting. Like all other Ixodinæ, both ticks pass through three active life stages—larval, nymphal, and adult. Preparatory to the transformation from one stage to the next, the feeding of the tick is suspended; then, after an interval of variable duration, the old skin is ruptured, the tick crawls from it, and very soon seeks to renew its attack. The cattle tick normally stays on one animal throughout the various changes. It remains attached to the skin by its rostrum after it suspends feeding both as a larva and as a nymph, and hence, on the completion of the moult which follows, it has only to reattach itself to the animal by its new mouth part, an act which it performs almost at once. The dog tick, on the other hand, perhaps because it infests an animal more energetic than an ox in locating and destroying skin parasites, has the habit of invariably loosening its hold and seeking shelter in the ground as soon as it ceases feeding before a moult; and in consequence it has the trouble of finding a host three times in its life instead of once, as with the cattle tick, and more often than otherwise a different dog is found each time. The females of both species fall to the ground when they are replete with blood, and perish soon after laying their eggs. The cattle tick, thus debarred from attacking more than one animal, can only transmit any pathogenic organism it may imbibe by passing it on to its progeny, a process which requires many weeks or months for its completion, whilst the dog tick can, perchance, transmit an organism imbibed in the larval or nymphal feeding stage to another animal in its own subsequent attack a few weeks later.

RED WATER, HÆMO-ALBUMINURIA (SIMONDS); BLACK WATER, BLOODY URINE, HÆMATURIA (GAMGEE); MUIR-ILL, ETC.

A disease of the bovine tribe characterised by the emission of red-chocolate or black urine, containing albumen and the colouring matter of the blood in a broken-down or disintegrated condition. The disease is associated with great prostration,

febrile excitement, palpitation of the heart, a double, dicrotonous, or trembling pulse, pallor of the mucous membranes, and diarrhœa, succeeded by obstinate constipation of the bowels.

*Etiology.*—The so-called red water in cattle of the older writers may be classed under the heading of Piroplasma. The protozoan organisms have now been demonstrated as the cause of red water in almost every country, and the old theories of acorns and dead leaves, &c., being the cause are no longer tenable. Such substances when used as a diet may no doubt cause dietetic diseases, and even grave symptoms of poisoning, but red water as described may, as a rule, be taken as piroplasma.

The various forms of piroplasma or pyrosoma infection are manifested differently in the various animals, and each variety of parasites is specific for each species of animal—i.e., the variety affecting the horse will not be pathogenic to the dog, &c. In the horse, the disease is usually described as bilious fever, and is due to the *Piroplasma equi*; in cattle it is red water, due to *Piroplasma bigeminum*; in sheep it is due to *Piroplasma ovis*; in the dog it is malignant jaundice, due to *Piroplasma canis*.

The organism may be always found in the red corpuscles of affected animals in a greater or lesser degree, according to the severity of the attack. It is the same as has already been described in connection with Texas fever. In fact, the same disease is of universal distribution. In England and India it is comparatively benign, but in some parts of America and in South Africa it assumes very acute and fatal conditions. No doubt this is the result of a natural immunity. In all cases the intermediary agent is the tick, the varieties being in England, *Ixodes ricinus*; in Australia, *Boophilus Australis*; in India, *Boophilus Australis* and *Hyalomma Egyptium*; in America, *Boophilus* or *Rhipicephalus annulatus*.

The symptoms have already been described in connection with Texas fever, and the treatment and pathology is the same.

In the horse the leading symptom is the acute bilious fever (see p. 534).

In dogs the disease takes the form of a rapidly fatal malig-

nant jaundice (see p. 534). There is a particularly virulent variety of piroplasma in cattle in South Africa, known as heart water. It is so called from the characteristic symptoms—*i.e.*, the presence of acute exudative pericarditis. The course is rapid, and death occurs quickly. The intermediary tick is the Bont tick, *Hæmaphysalis Leachii*.

Another fatal piroplasma is the East Coast fever of South Africa, the organisms of which assume a bacillary form, and death is usually certain and rapid. This form closely resembles the tropical variety seen in India, when the organisms infecting the red corpuscles are very small and bacillary in shape.

Red water prevails among calves, stirks, oxen, and bulls, and extensive experience has shown that impoverished pastures, heathy moors, and woody districts are subject to have the cattle grazing upon them affected with the disease in an enzootic form, during some seasons.

On the Continent the disease has been recognised as connected with damp lands and wet seasons. It is seen always on pasture lands, and never in stall-fed animals.

The *post mortem* appearances indicate a condition of anæmia. The subcutaneous tissues are white; the blood-vessels and cavities of the heart are empty, leading one to suppose that the animal had been bled to death; ecchymoses are seen on the serous membranes, and on the lower surfaces of the kidneys. In no case are there indications of nephritis, the kidneys, on the contrary, being blanched, and the uriniferous tubes enlarged, evidently dilated with a viscid fluid. The rectum generally contains fæces covered with a dark-coloured mucus; in nearly all cases the liver is darker than is natural; the gall bladder distended sometimes with a dark viscid, sometimes a thin yellow, bile. The contents of the lacteals and thoracic duct are occasionally found to be of a dark red or brown colour; but the most constant pathological conditions, in addition to the anæmia, are softening and engorgement of the liver, and fulness of the gall bladder.

TICK PARALYSIS AFFECTING SHEEP AND LAMBS: AN INQUIRY  
BY VETERINARY SURGEON BORTHWICK, CAPE OF GOOD  
HOPE GOVERNMENT REPORT, NOVEMBER 3, 1904.

"This 'tick paralysis' is met with over a large area of the Colony, and has been recognised by the sheep farmers for many years, and unanimously attributed by them to a certain tick now identified by Mr. Lounsbury as the *Ixodus vilosus*.

"As it was more than usually prevalent during the last winter on several farms on the Fish River Rand, Mr. Borthwick was instructed to proceed to that district and conduct an inquiry into its nature and cause. The following is his report:

"The farms on the Fish River Rand on which tick paralysis is more or less prevalent are: Hopewell, Hartmans Kraal, Groenfontein, Thornkloof Rudsdal, Kagasomdt, Brakfontein, Olivefontein and Waterfall; it also occurs on one farm in the Somerset East district. There are probably a good many more farms on which the disease is prevalent, but I have not heard of them.

"*Time of Year.*—It is during the months of May, June and July that the disease is most prevalent, some farmers stating that it only occurs every second year, but on the Somerset farm it has been appearing every year for the last three years.

"*Condition of Veldt.*—It is seldom that cases are found when sheep are running on short veldt, but in the kloofs when the veldt is long and there is some bush the disease becomes exceedingly prevalent.

"*Tick.*—The tick which is credited with producing the disease is the *Ixodus pilosus* (Lounsbury); the tick attaches itself principally on the inside of the thighs, behind the elbows and between the lower jaws. The ticks are also found on sheep which are not paralyzed.

"*Symptoms.*—In grazing over the veldt a sheep will be noticed to lag behind the others and then lie down, but on the approach of the herd it will get up and walk after the flock; it soon, however, lies down again, and seems to have more difficulty in getting up after each rest. Shortly afterwards

the sheep will lie down, and on the herd's approach, will not even attempt to get up, and shows no sign of fear or excitement; if lifted up, it may walk a short distance with an unsteady gait, but soon falls down, and if the lifting up is persisted in the sheep soon becomes unable to stand. From the time that the animal is first noticed to be infected until completely paralyzed may only occupy six hours. After the sheep is completely paralyzed, it remains as if asleep for from twenty-four to forty-eight hours, when in the majority of cases it makes a rapid recovery. The temperature is normal.

“ ‘The above symptoms are those of a case which has been placed, while in the early stages of the disease, in a shed and left undisturbed. Very few die if so treated, but the deaths from the disease in the ordinary way are fairly heavy; when the paralyzed sheep is left out at night the ever watchful jackal usually plays the last act, or the sheep, when still able to walk a little, may fall into a sluit and die.

“ ‘The number of ticks does not seem to influence the severity of the case, two ticks inducing as bad a case as fifteen; but if the ticks are removed immediately the sheep is observed to be sick the recovery is more rapid—within twelve hours in many cases recovery is complete.

“ ‘The few *post mortem* examinations which I was able to make did not show any noticeable change in the organs. Veterinary Surgeon Spreull made inoculations with the blood of affected sheep into healthy sheep, but the results were negative.

“ ‘To ascertain if dipping had any preventive action, I had a flock of eighty-eight grazed in a kloof where the disease was prevalent. Twenty out of the number were left undipped, the remainder being dipped in Cooper's dip. The result was that within three weeks six out of the twenty became paralyzed, three of which died, and not a single case occurred amongst the dipped sheep. On the same farm two flocks of 700 and 650 in which the disease was prevalent were also dipped, no cases occurring subsequently.

“ ‘On Hartmans Kraal, during the month of June, Mr. Innes had between sixty and seventy sick, but after dipping the cases ceased; the lamb, however, after being kept in the kraal

for a fortnight, on being turned into the veldt, began to sicken, but were dipped, with the same result.

“‘I had arranged to try the effect of dosing with Cooper’s dip, but that experiment fell through.’

“*Inoculation Experiments.*

“Mr. Spreull took some blood from an affected sheep, forwarded to him by Mr. Borthwick, and inoculated two healthy sheep. These sheep had been already rendered immune to malarial catarrhal fever. The inoculation failed to induce any febrile reaction or indication of constitutional disturbance.

“A second inoculation was made with blood obtained from a yearling hamel, which was brought to the station by Mr. Borthwick on his return. This hamel was in a somnolent condition on its arrival. The two sheep which were selected for this inoculation had not been used previously for any experiment, and each of them gave a distinct febrile reaction on the eighth day after inoculation. Blood-smears were taken from one of these on the tenth day and microscopically examined, but no micro-organisms were found. These two sheep were subsequently inoculated with blood from a sheep affected with malarial catarrhal fever, and both became affected. (It would appear from these two inoculation tests, that an attack of malarial catarrhal fever confers a certain amount of resistance to this tick paralysis, but that an inoculation with blood from a sheep suffering from tick paralysis gives no immunity to malarial catarrhal fever. But these experiments will require to be multiplied before any definite conclusions can be drawn.)

“*Post Mortem Appearances.*—The first case was a yearling Merino ewe, in poor condition; the heart-sac contained a little serous fluid, blood clotted fairly well in heart and large vessels; the lungs were congested, liver and kidneys a little congested, spleen normal, the small intestines inflamed throughout, stomachs and large intestines healthy. The second case was a yearling hamel; all the organs appeared healthy; there was a slight amount of watery effusion in the heart-sac, and an increased amount of fluid in the spinal canal. The flesh looked sodden from prolonged lying, but was not jaundiced; the blood was bright red and clotted firmly.

“Blood-smears from both these affected sheep were examined by the microscope, but no micro-organisms were detected.”

## CHAPTER XLV.

### ACTINOMYCOSIS.

THE new formations which result from the invasion of the tissues by the actinomyces or ray fungus were classed as parasitic tumours. These growths are found usually in three situations—namely, the tongue, the maxillary bones, especially the inferior maxilla, and the subcutaneous tissue of the throat (wens). Exceptionally, the udder, the lung, or the intestines are affected. Histologically examined, the lesion is seen to result from the development of granulation tissue, the subsequent changes in which vary with the site. In the tongue a dense fibrous tissue results, with scattered foci of small round cells, pretty closely resembling tubercles. In these the actinomycotic colonies are found.

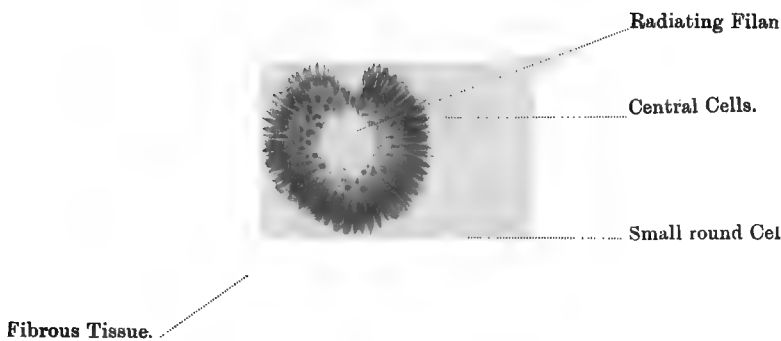
In the maxilla the granulation tissue at first formed opens up the bone, giving rise to absorption and new formation of osseous tissue (rarefying osteitis). Suppuration takes place at numerous points, and, the pus ulcerating its way to the exterior, gives rise to fistulæ.

In wens, which are the common form of actinomycosis among cattle in some English counties, the granulation tissue breaks down, and there is formed a dense capsule of fibrous tissue containing pus, in which the colonies of the organism may be seen as yellowish grains.

Stained by Gram's method, and examined microscopically, the actinomyces is seen to consist of a central homogenous part, which takes the counter-stain and radiating filaments, with clubbed ends stained purple, the whole recalling the capitulum of a daisy.

*Animals affected.*—The disease is rare in animals other than





× 500.



× 250.

W. O. W.

TWO VIEWS OF THE RAY FUNGUS (ACTINOMYCES)  
UNDER DIFFERENT MICROSCOPIC POWERS.

STAINED WITH HENEAGE GIBBS'S TUBERCLE BACILLUS STAIN, AND MOUNTED



the ox, but neither horses, sheep, nor pigs are immune. In the last animals the udder is usually the site of the disease.

*Treatment.*—Iodine dissolved in water by iodide of potassium may be given daily, and has proved satisfactory when the bones are not greatly involved.

Wens are treated by evacuating the contents, and irrigating the cavity with a strong solution of iodine, which may also be given internally.

Tumours simulating those produced by actinomyces, but in reality due to another organism—the *Botryomyces* or *Discomyces equi* (Rivolta)—are found affecting the horse in “scirrhus cord,” and in tumours about the shoulder and legs.

They consist of dense fibrous tissue, in which are suppurating centres containing grape-shaped colonies of the organism, which some observers, however, consider to be nothing more than zooglea masses of *Staphylococcus pyogenes*. The treatment is similar to that indicated for wens.

## CHAPTER XLVI.

### SPORADIC DISEASES.

#### RHEUMATISM.

##### ACUTE RHEUMATISM, OR RHEUMATIC FEVER.

*Definition.*—An inflammation of the fibrous structures of the joints, tendons, ligaments, thecæ of muscles, or of the heart and closed cavities, due to a specific condition of the blood, and accompanied by fever, stiffness, and lameness. The inflammation is metastatic or erratic in its character, disappearing from one part of the body to reappear in another, without any appreciable cause. The inflamed parts are generally swollen and hard, but in some instances no swelling can be detected.

Characteristic of rheumatic inflammation both in man and the lower animals are—(1.) The rarity of suppuration. This process may and does sometimes occur, especially in horned cattle, from inflammation of the superficial coverings of the diseased tissues, caused by external bruising, or the application of powerful external irritants. (2.) The occurrence of cardiac complications. (3.) A natural predisposition or diathesis, which predisposes to the malady without any ostensible cause. The form of the disorder in this instance is of a less acute nature, but may become acute by the addition of any trivial cause of disease.

*Etiology.*—The application of cold; damp; or it may result from other diseases, as epizootic diseases, contagious eczema, or from a natural predisposition without apparent cause.

*Semiology.*—The symptoms of rheumatic fever are as follows:—Sudden lameness, with or without swelling of some particular articulation, such as the stifle, hock, or fetlock joints; the flexor tendons, immediately below the knee or hock, in the sesamoid bursa, the thecæ of the muscles of the loins and quarters, or of those of the thoracic walls, constituting pleurodynia. The lameness may be preceded by some febrile disturbance or a *malaisé* condition, expressed by yawning, dulness, or dejection.

The lameness often disappears from one part of the body and suddenly reappears in another. Very often the lameness is symmetrical, that is to say, it will be due to inflammation of the same joints in both legs, say in two stifle or in two hock joints. The fever is acute and sthenic when it is not preceded by some epizootic disease; the pulse is hard and unyielding; the mouth hot and dry. The urinary secretions are impaired and altered; the urine, which in health, when tested with litmus paper, gives an alkaline reaction in the herbivora, is neutral, or more or less acid, and if microscopically examined is found to be loaded with hippurates of soda and ammonia, and hippuric acid.

There is generally some degree of costiveness, and if blood be drawn, the coagulum will be firm and large; indeed, in no disease is there such a rapid and sensible increase of fibrin in the blood as in acute rheumatism—in some cases as much as 10 parts in 1000 of blood have been found. The swellings of the affected parts quickly assume a hardness due to exudation, but, as already stated, suppuration rarely occurs. The elevation of temperature is sometimes very great—104°, 105°, or 106° F.; when over 105° F. it is always indicative of great danger. I have known it as high as 109° F. in a cow.

#### CHRONIC RHEUMATISM.

The symptoms of this form are mere modifications of those of the former, except that fever may be entirely absent. It is, however, much more persistent, less metastatic, and leads to alterations of structure, consisting of ulceration of articular cartilage, eburnation of the bones, and the formation of osteophytes, much more commonly than the acute, an attack of which may leave the animal quite well at its termination. Sometimes, however, the acute degenerates into the chronic, and an animal subject to the chronic is very often attacked by the acute form. In chronic rheumatism not only are the white fibrous structures altered, but the bones in various parts of the body may become subject to various pathological changes; tumours or bony excrescences form on the bones of the pelvis, spinal column, and in the fringes of the synovial membranes; distortions also occur. I have seen the neck twisted by large bony tumours on the cervical vertebræ, arising from this cause; also enlargement and ankylosis of the joints, ulceration of articular cartilages, particularly of the navicular bursa, eburnation of the exposed bony extremities, and

ossification of the walls of the heart. The soft structures above and below the affected joints in horned cattle sometimes swell, suppurate, and continue to discharge pus for a lengthened period, the animal rapidly wasting, and finally becoming worthless.

Both in the acute and chronic forms the pericardium, endocardium, and cardiac valves may become inflamed. This complication, however, is of much more frequent occurrence during an acute attack. The pulse becomes jerky and wiry, the heart's action short, sharp, and angry, the cardiac impulse is often wanting, the pulse intermittent, and a to-and-fro sound accompanies the heart's movements.

#### PATHOLOGY.

What the true nature of the so-called rheumatic poison really is, pathologists are not agreed upon, some asserting that it is lactic acid, whilst others are not satisfied on this point. It is certain, however, that the urine both in man and in the lower animals assumes an acidity which is quite unnatural; but the acid found in it is not the lactic, but the uric and its salts in man, and the hippuric and its salts in the herbivora. The suggestion that rheumatism is due to the presence of an excess of lactic acid originated with Dr. Prout. The lactic acid originates by the conversion of the starch of the food into lactic acid, which then combines with oxygen to form carbonic acid and water; and anything which interferes with this change may lead to the accumulation of the acid in the system. Dr. Garrod, however, found the serum of the blood distinctly alkaline in thirty-five cases of rheumatism in the human being.

In every case of rheumatism met with by myself, and where an examination of the serum has been made, it has been found alkaline. The only experiments which go at all to establish the connection of lactic acid with rheumatism are those of Dr. W. B. Richardson, who injected lactic acid into the peritoneal cavity of dogs, and found that it caused death, not by inducing peritonitis, but peri and endocarditis, with thickening of the cardiac valves; the *post mortem* examination showing a red thickened state of the mitral valve, with fibrous bead-like deposits on its surface; the endocardium lining the left ventricle intensely inflamed, resembling bright red velvet, and the aortic valve of a deep red colour, and its borders thickened

and everted; the pericardium red, injected, and covered with lymph.

Dr. Fuller believes the poison of rheumatism to be identical with some natural excretion of the skin. The researches of Dr. Parkes lead him to conclude that the disease is due to some substance rich in sulphur; whilst other writers and observers look upon rheumatism as simply an inflammation of the fibrous and serous membranes, the predisposition to which arises from deficiency of healthy tone in these textures, rendering them liable to be inflamed by the influence of a variable temperature. The character of rheumatism, more especially its metastatic tendency, and the symmetrical development of its symptoms, tend to show that an alteration in the blood exists; the only constant alteration is, however, the great increase of fibrin. We are thus led to the conclusion that, whatever the morbid material may be, it is one formed within the economy, and due either to a faulty secondary digestion and assimilation, or to an abnormal metamorphosis of tissue and the retention of the products of such metamorphosis within the animal body. In man copious perspiration is a characteristic sign of rheumatism, and it is to be noticed that this perspiration has an acid or sour smell. In the lower animals perspiration may be copious, but sourness of smell is not appreciable.

In rheumatic endocarditis in man, the left side of the heart is only affected, as a general rule; and it has been inferred by Dr. Richardson that the chemical change whereby the morbid matter of acute rheumatism is produced, is completed in the pulmonary circulation; that when the blood in the pulmonary capillaries is exposed to the air, the acid quality of the poison is produced; that the poison thus formed is carried by the arterial circulation to be disposed of by decomposition or elimination, or both; and that it does not return as an acid by the veins, but simply as a product which admits of retransformation in the pulmonic circuit into the acid state.

#### TREATMENT.

*In the Horse.*—A gentle laxative may be given with advantage, and there is no such objection to an aloetic purge as in some of the diseases which have been enumerated. If the pain be excessive, opium may be combined with the purgative, and

repeated once or twice a day. To prevent its constipating action on the bowels, the opium may be combined with calomel, but calomel should not be given until the bowels are "set" after the purgative. If the pain is not excessive, aconite and the nitrate of potash are more suitable than opium, and in all cases the nitrate of potash is to be freely administered, in order to alter the character of the blood, to act upon the excretory organs, and to influence the vascular system. In combination with the nitrate, colchicum has a favourable effect, and in cases where debility is not great it should be substituted for the aconite about the third or fourth day. The administration of salicylate of soda is found to be of great benefit in place of the nitrate of potash, in fact, nowadays it is preferred. If the sufferer be strong, young, and in good condition, a moderate bleeding is usually prescribed; as a rule, however, the conditions do not warrant the abstraction of blood. Indeed, it is now generally agreed that although bleeding, by relieving congestion, may give temporary relief, it enhances future suffering and danger, by increasing the irritability of the heart, and consequently predisposing it to rheumatic inflammation.

*Local treatment.*—If the pain in the inflamed parts be very great, it may be advisable to endeavour to relieve it by fomentations, and the application of a liniment containing some of the extract of belladonna, opium, or aconite; but valuable time should not be wasted if these afford no relief, and blisters are to be freely applied to or near the inflamed parts. The treatment by blistering is called that of *elimination*, and those who advocate it most strongly use it only. According to Dr. Herbert Davies, Physician to the London Hospital, the action of blisters, by promoting a serous discharge from the blistered surface, affords a ready means of exit to the rheumatic virus.

The success of this treatment depends upon the amount of *vesication* produced by the blister; and in order to promote the free flow of serum, the blistered parts are to be subsequently covered by linseed-meal poultices. According to Dr. Davies, the blister treatment causes a speedy diminution in the frequency of the pulse, rapid subsidence of the joint affection, and lessens the liability to cardiac inflammation. Within twenty-four hours after the removal of the blister, the urine is stated to become alkaline in reaction.



Dr. Davies does not apply the blister upon, but near to the inflamed part. I can speak with great confidence of the benefit of treatment by blistering in the lower animals; the blisters are, however, quite as effectual when applied to the inflamed parts.

*In the Ox.*—The same external treatment is applicable as in the horse; the nitrate of potash may, however, be given in larger doses, and the bowels acted upon by the sulphate of magnesia or soda instead of aloes. Cows will often, when suffering from rheumatic fever, refuse to rise from the recumbent posture. In such cases repeated change of bedding is absolutely necessary; great cleanliness, dryness, and comfort must be secured, and the inflamed joints wrapped in dry flannel, in order to prevent bruising and the occurrence of the suppurative process.

With regard to cardiac complications, I think the cow is not quite so liable to suffer as the horse: this is, however, a matter of opinion merely.

If suppuration occur in or near to an articulation, it is best at all times to put the ox or cow out of its sufferings, as recovery seldom or never takes place.

The diet in all cases, if the animal be not already debilitated by previous disease or other cause, must at first be light and easy of digestion, as mashes and hay, or green food, if in season; as the fever subsides, stronger food may be cautiously allowed; and if depression be a marked symptom, cinchona and ammonia are to be prescribed in suitable doses.

Continental writers describe articular rheumatism as a febrile infectious disease, most frequently affecting horned cattle, and often due to abortion or non-delivery; and they consider that the absorption of septic products of the uterus is the sole cause of articular rheumatism. I cannot agree with this view, and the writers have evidently confounded rheumatism with septic conditions sometimes witnessed immediately subsequent to parturition, abortion, or co-existent with retention of foetal membranes or of a dead foetus.

The treatment of rheumatism, according to Continental writers, is a very complicated affair, consisting of irritating and stimulating frictions to the affected regions, the induction of diaphoresis, massage; the administration of salol, naphthol, antipyrine, laxatives, tartar emetic, tartar colchicum, pilocarpine, &c. The reader can have his choice; the author has tried a few of the remedies, and found them inoperative unless for harm.

## CHAPTER XLVII.

### SPORADIC DISEASES—*continued.*

#### DIETETIC DISEASES.

IN order that the health of the animal body may be duly maintained, it is necessary that the various kinds of food should be sufficient in quantity and undeteriorated in quality. It is also necessary that the solid food contain at least four classes of constituents, each of which plays an important part in supplying the various wants of the economy, either as to supplying materials for tissue formation or for force. These constituents are—(1.) Nitrogen, nitrogenous or proteid food, which seems most fitted to nourish muscular and other albuminoid tissues; on this account the substance containing it has been called plastic or nitrogenous food. Substances defective in nitrogen, and having an excess of carbon or hydrogen, are called (2.) carbohydrates or starchy foods, consisting of elements which easily break up into carbonic acid and water, supply materials for combustion, and thus assist in maintaining animal heat and other energies required by the system. (3.) Fats, or hydrocarbons, like the carbohydrates, have no nitrogen, require a great supply of oxygen, and evolve a large amount of animal heat. Food must also contain (4.) saline materials, in order that the solid structures of the body be built and maintained in health; the saline constituents also aid in the processes of assimilation and elimination, of carrying new materials into the system and old materials out of it. If these constituents are absent, or are present in undue proportions, health cannot be maintained, and common experience has taught us that all animals are kept in the best health when fed on a mixture of foods.

Although chemical analyses have enabled us to know the quantities of nitrogen, carbon, &c. contained in the food, it does not follow (as General Fitzwygram justly remarks) “that the food which possesses these constituents in the greatest abundance

will produce the most beneficial results. Digestibility, readiness of assimilation, absence of unduly heating properties, and many other qualities, are needed in order to make a substance possessing the necessary ingredients available as food. Chemistry is a valuable but not an infallible guide, and its indications require to be tempered by the test of experience."

Professor Dick said that a horse may be kept without work, but taking a little exercise, in fair condition on 12 lbs. of hay and 5 lbs. of oats per day; but if a good amount of work is to be got out of it, the horse should have 14 lbs. hay, 12 lbs. oats, and 2 lbs. beans. In this diet of the horse at rest there are 29·2 ounces of flesh-formers, and 150 ounces of heat-givers and fat-makers; and in the work diet there are 251 ounces of heat-givers and 59·1 ounces of nitrogenous constituents.

It is not my intention to enter into a lengthy discussion on the above subject. My experience leads me to conclude that the scale laid down by Professor Dick is too limited; that it is necessary to give to a horse at rest several pounds more corn per day in order to keep it in good health. Every-day experience teaches us that no horse can be kept in health without exercise, and no horse taking sufficient exercise to keep it in health can maintain its vigour and flesh on 5 lbs. of oats per day.

I am also of opinion that all horses at work should have a mixture of oats, beans, and hay; say 14 lbs. oats, and 2 lbs. to 4 lbs. of beans per day. I find this to be the cheapest and best of food. It must be given either whole or crushed, dry, and it is advantageous to mix it with chopped hay. The quantity, however, must vary with the size of the animal.

The corn and hay must also be of good quality, sweet, free from mildew, well harvested, old, and dry.

For further information on the subject of feeding, the reader is referred to Sellar and Stephen's *Physiology at the Farm*; General Sir F. Fitzwygram's *Horses and Stables*; Smith's *Veterinary Hygiene*; Low, Playfair, Voelcker, and others.

*Effects of over-feeding.*—Too much non-nitrogenous food favours the development of fatness and obesity. This is well seen in pet dogs and cats fed on cream, sugar, and tit-bits of various kinds. These animals die before half their days are over from fatty infiltration and degeneration of the heart, liver, and other organs. Lions, tigers, and other carnivora kept in menageries

also suffer and die from fatty degenerations, and old carriage and other favourite horses die from the same cause, when pampered and fed with carbonaceous food.

Accumulations of the flesh-forming or nitrogenous elements in the blood, and their non-elimination, cause the development of many blood diseases, as has been already, and will be further shown. For example, over-feeding on linseed cake induces a septic condition of the blood. Analyses of oil-cake have shown that it contains over 22 per cent. of nitrogenous materials, whilst dried wheat contains only 12·47 per cent. of albuminous compounds. Again, decorticated cotton cake contains a much larger per-centage of flesh-forming matters than even linseed cake, and the proportion of oil is higher than in the best linseed cake. The following are the analyses of the two cakes :—

DECORTICATED COTTON CAKE.	OIL-CAKE.
<i>Average analysis of seven Samples.</i> (VOELCKER.)	(PROFESSOR JOHNSTON.)
Water, . . . . . 9·28	Water, . . . . . 10·05
Oil, . . . . . 16·05	Mucilage, . . . . . 39·10
Albuminous compounds, or flesh-forming matters, . . . . 41·25	Albumen and gluten, . . . 22·14
Gum, mucilage, and digestible fibre (heat-producing substances), 16·45	Oil, . . . . . 11·93
Celluline, indigestible fibre, . 8·92	Husk, . . . . . 9·53
Mineral matters, ash, . . . 8·05	Ash and sand, . . . . . 7·25
<hr/> 100·00 <hr/>	<hr/> 100·00 <hr/>

The nitrogen of the food is not all assimilated in the system, and a large portion passes away with the excreta: on this account the dung produced by cake-fed stock is particularly valuable. Should anything occur to interfere with the functions of the excretory organs, the nitrogenous compounds accumulate in the system, and there set up a variety of diseases more or less grave and important. It will thus be seen that great care and discrimination are to be exercised in feeding stock upon highly nutritious food; and provided the feeder has an eye more to the maintenance of the health of his stock than to the richness of his manure, he will bear this in mind.

Whilst over-feeding leads to the development of disease, deficiency of food leads to no less grave results; and this

deficiency may relate both to quantity and quality. "A deficiency of food," says Dr. Letheby, "especially of the nitrogenous part, quickly leads to the breaking up of the animal frame. Plague, pestilence, and famine are associated with each other in the public mind, and the records of every country show how closely they are related." It is stated that in cases of very gradual starvation an urgent feeling of hunger is not a prominent symptom, and even when it exists at first, it usually soon diminishes, and is succeeded by a feeling of exhaustion and faintness, and even a loathing of food, if abstinence has been long protracted. —(R. B. HOLLAND.)

Whilst it is essential to health that food should be sufficient, but not over-abundant in quantity, and that its quality should be so regulated as to supply all the wants of the economy, it must be confessed that the lower animals will live for a very long period on very common fare, provided it be sufficient in quantity, and they are not exposed to extreme cold; but to have health, energy, and condition, both food and water must be good in quality.

A dietetic disease may be defined to be a morbid condition of the body, brought about by food or water deteriorated in quality, insufficient or over-abundant in quantity, or containing some ingredient directly poisonous or injurious to the animal economy.

## CHAPTER XLVIII.

### SPORADIC DISEASES—*continued.*

#### DIETETIC DISEASES—*continued.*

DIABETES INSIPIDUS, POLYURIA, HYDRURIA—  
AZOTURIA, OXALURIA — ASTHMA, BROKEN  
WIND, &c.

#### DIABETES INSIPIDUS, POLYURIA, HYDRURIA.

A DISEASE characterised by great thirst, excessive discharge of urine, rapid emaciation, languor, and debility. In the majority of instances it is caused by deteriorated food, but in some cases it seems to be due to some constitutional cause, produced through derangement of the assimilative functions, either in the digestive canal, the solid organs, or the blood. When induced by no traceable cause, and where change of diet has no effect in checking it, the disease is generally premonitory to farcy or glanders, and is symptomatic of a breaking up of the tissues of the body.

*Etiology.*—Diabetes is induced by two kinds of causes, namely, intrinsic and extrinsic. The intrinsic causes are those originating in defective assimilation, or rapid tissue metamorphosis, owing to the actual presence of the glanders poison, or to a condition of the system tending to its development; the polyuria being induced by the action upon the kidneys of those constituents, products of tissue change, which are naturally eliminated by them. In some instances diabetes results from indigestion, disappearing when the digestive apparatus is restored to its normal condition. It also accompanies other diseases where the digestive process is defective.

The extrinsic causes are to be found in the food which the animal consumes; I am not aware that it is ever induced by the water it drinks. Dark-coloured, highly-heated hay generally

causes much thirst and diuresis, probably containing some ingredient which acts as a stimulant to the kidneys, but it does not induce diabetes so readily as hay that is musty, oats or beans which have been musty or damp, even if kiln-dried, and bran having a peculiar greasy odour, more especially foreign bran.

Diabetes sometimes occurs as an enzootic disease—not due to any atmospheric influence, but following a bad harvest, and caused by damaged food. All writers agree in condemning kiln-dried oats. I am of opinion that these, if of fair quality when put on the kiln, are not so hurtful as is generally supposed, and that they are much more likely to cause disease if given when damp or musty.

In a stud of horses where glanders prevails, the slightest error in feeding the apparently healthy animals will often bring on profuse diuresis. In one stud under my care, a supper of warm boiled food was sure to be succeeded by several animals being attacked by inordinate thirst and diuresis. Habitual feeding on boiled food is a very common cause, and although animals so fed may look moderately sleek, and lay on fat, they are incapable of performing the same amount of work as others fed on similar food given uncooked.

*Semiology and Pathology.*—The diagnostic signs are excessive thirst and profuse urination: in addition to these there is failing of the appetite. Some teachers say the appetite is increased; I have never known this in the horse, but in the dog there is often a craving for flesh. The visible mucous membranes are pale or sometimes rusty yellow; the skin is harsh and the coat unhealthy looking; the animal is debilitated and rapidly loses flesh. The pulse is often slower than natural, sometimes quicker, and always atonic. The mouth has a sour odour; the horse prefers unclean food and water, and is fond of licking the walls and manger. The urine is of a very pale colour, sometimes as clear as clean water, and has a specific gravity little higher than distilled water, the specific gravity of which is 100, whilst that of diabetic urine is from 100·2 to 100·3, and that of healthy urine in the horse ranges from 100·30 to 100·50. According to Lassaigne, the diabetic urine of the horse contains—water 98·0; urea, benzoate of soda, acetate of potash, acetate of lime, chloride of sodium, and free acetic acid, 1·5; mucus and sulphate of lime, 0·5; and differs from healthy urine

in (1.) being more watery ; (2.) containing acetic acid, and in being free from earthy carbonates.

The diabetes mellitus differs from the insipidus, from the circumstance that the urine contains sugar. I am not aware that this form is ever seen in the herbivora ; but I have repeatedly met with it in dogs, and in almost every instance the creatures had been fed for a length of time on boiled liver. M. U. Leblanc reports a case in a dog and another in a monkey.— (See GAMGEE'S *Domestic Animals*.)

*Therapeutics*.—Investigate carefully the quality and quantity of the food, which in all cases, whether it be apparently good or indifferent, ought to be changed. If the horse is fed on oats and hay, try a change to beans and a fresh sample of hay ; if fed mostly on beans, change to oats. The bran and other fodder must also be changed, as any of these articles may contain some undetectable material which operates unfavourably on the economy, some constituent which acts as a constant irritant upon the body and kidneys. It is advisable to give an aperient, and to restrict the diet to hay and mashes for some days. Much relief will be afforded to the animal by giving the bicarbonate of soda in the food or water. I prefer this to chalk, a remedy held in well-deserved esteem.

When the aperient has operated, iodine, either alone or in combination with sulphate of iron, is to be administered, commencing with two-drachm doses twice a day, and diminishing the dose as the thirst disappears.

Iodine was first prescribed for diabetes by Professor Dick, who discovered, whilst experimenting with it in the treatment of glanders, that it had the effect of allaying the sense of thirst in a most remarkable manner, and ever since then it has been held to possess specific powers in this affection. There is no doubt whatever as to its therapeutic value, for not only does the thirst diminish, but the appetite improves, and all the other symptoms disappear as soon as the system is charged with the iodine.

It is usual to mix clay with the horse's drinking water ; if soda bicarbonate is prescribed, there is no need to give dirty when clean water can be obtained, and it is cruel to restrict the water in quantity to any great extent. The horse must rest, and be carefully attended to for several days.



In the dog the disease is fatal. I have tried various remedies, such as feeding on milk or on flesh entirely; creosote, opium, astringents, the bromide of potassium, as well as the iodine treatment, and have seen no permanent benefit from any of them. Both iodine, or iodide of potassium, and the bromide moderate the symptoms for a time, but usually the benefit has been of short duration—a cough has come on, rapid emaciation, decay, and death.

*Post mortem appearances.*—Both in horses and dogs, pallor of the organs and tissues of the body generally; in dogs, enlargement of the liver and caseous tumours in the lungs—the latter being probably accidental complications.

#### AZOTURIA.

Under the term of azoturia, or nitrogenous urine, I intend to describe a disease to which attention was drawn in this country by Haycock, who named it, at the suggestion of Mr. James Moore, V.S., London, "Hysteria." In Gamgee's *Domestic Animals*, the same malady is described as "enzootic hæmaturia of horses"—"*schwaze harwindi*" of the Germans, who state that it occurs in horses, and very rarely in mares. Mr. Haycock, on the contrary, says it occurs in mares only, and for this reason has called it hysteria. I have, however, seen it both in mares and horses, and have satisfied myself that in no instance is there blood in the urine, but that the dark coffee colour is due to the presence of urea and colouring matter.

The symptoms are well described by Haycock, who says—"In all cases of this nature which I have treated the disease commenced very suddenly. They [the subjects thereof] began to exhibit an unusual degree of restlessness, to perspire profusely, which symptoms or states were speedily succeeded by a disposition to lie down; by great sluggishness, loss of motor power in the hind limbs; violent spasm of the large muscles of the loins and hind quarters (the gluteal muscles were excessively cramped), and the shoulders; the pulse in two of them rose from sixty to eighty beats per minute, and the respirations were greatly increased; they made several efforts to rise upon their feet, but from the total loss of all motor power in the hind limbs, they were unable to do so; two out of the three every now and then strained violently, and ejected, *per vaginam*, excessive quantities of coffee-coloured urine, which consisted principally

of blood; the perspiration was excessive also. In two cases the more violent symptoms subsided for a time; they became cool in the skin, and partook of food and water, but were totally unable to rise. This improvement did not prove of any long duration; they commenced struggling again with renewed violence with the fore limbs, and continued to do so until they died." Gamgee gives the symptoms of the disease as it occurs in Germany as follows:—"Stiff gait, weakness of the hind quarters, frequent pulse, redness of the visible mucous membranes, anxious expression of countenance, and sweating; a remarkable swelling of great firmness occurs over the loins and hips, and there is a copious discharge of urine of a very dark red or brown colour. Great difficulty of breathing ensues, tetanic symptoms supervene, and death."

The two descriptions agree in most particulars; both contain the diagnostic symptoms of the malady, namely, the tonic spasm of the gluteal, lumbar, and some scapular muscles, and the profuse discharge of dark-coloured urine.

Mr. Haycock was of opinion that the disease had some connection with the period of oestrus, that it was the result of undue excitability, and he compares it with hysteria as described by Copland in his *Medical Dictionary*. Mr. Gamgee offers no hypothesis as to the nature of the malady. Professor Dick describes it as sprain of the *psoæ* muscles.

At first I was inclined to think, with Haycock, that it was a disease of the mare, but further experience led me to conclude differently, for I have as frequently seen it in the gelding as in the mare; and examinations of the urine, both chemical and microscopic, have convinced me that there is no blood passed by the kidneys, but immense quantities of urea and a less notable quantity of hippuric acid.

The pathology of the disease is, in my opinion, a hyper-nitrogenous condition of the blood and system generally, due to over-feeding and want of exercise; the excessive secretion of urine and excretion of urea being physiological results due to the presence of effete products—metamorphoses of nitrogenous food—in the blood. The history of every case that I have met with, and of all those recorded by Haycock, points to this. He says—"The first case which I treated, the animal had rested about a week, at the end of which time it was put

into harness upon a very hot day, and driven slowly for a distance of five miles, and was seized on the road. The second case also occurred after the mare had rested a week or a little more, when it was taken out of the stable on a very sultry afternoon, and galloped severely, and shortly afterwards was seized. The subject of the third case rested from Saturday morning to Monday morning, at which time it was put to its usual labour, and driven slowly for about a mile, and was seized ere it had got to the end of the journey, short as it was." The above corroborates my experience of every case of this malady in a most remarkable manner. Varying periods of rest were succeeded by an attack on the first journey, the animals always leaving their stables in higher spirits than usual, and giving rise to the remark, "He never looked better than when he first turned out; in fact we could scarcely hold him, he was so spirited." I have only met with one case that was attacked in the stable prior to some amount of exercise. It seems necessary that some degree of muscular exertion be performed, and the only way in which I can account for this is, that the blood before exercise contains a superabundant quantity of albumen unappropriated by the tissues, and that the exercise, by increasing the rapidity of the circulation and of the respiratory movements, induces a rapid oxidation of such superabundant albumen, whereby it is transformed into urea, hippuric acid, &c., with which the blood becomes overloaded, and the kidneys stimulated to excrete what is proving deleterious. Albumen is occasionally present in the urine, but this is by no means constant; its presence, however, points to an aggravated form of the disease, and is often prognostic of a fatal termination.

The presence of such effete material in the circulation provokes tonic spasms of the muscles, loss of motor power in the posterior, and sometimes, but rarely, in the anterior extremities, tetanic convulsions, and, finally, death by extreme muscular prostration, simulating motor paralysis. In every fatal case that I have observed, the spasms and convulsions have been succeeded by extreme muscular debility, the muscles contracting but feebly on the application of a stimulus, the heart and diaphragm partaking of this prostration, and the animal dying from asthenia and apnoea. In other cases the

animal has overcome the primary and violent symptoms, but has remained partly paralyzed in one or both hind extremities, a remarkable circumstance being that the vastus externus and internus, and the rectus femoris in some cases atrophying to such an extent as to show a hollow above the stifle, and to render the patient unable to sustain the weight of its body, so much so that it crouches when standing, and when walked, walks like a whipped dog.

*Post mortem* examination reveals the blood dark coloured, having an ammoniacal smell, and semi-fluid; congestion of the lungs; clots of dark blood on both sides of the heart; the bladder filled with dark or coffee-coloured urine, and sometimes softening of the liver and kidneys.

Congestion of the sheaths of the great gluteal nerves has been present in some cases, in which the spinal cord at the lumbar region has been found red, congested, and softened,

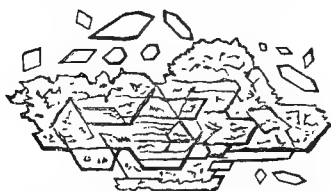


FIG. 56.—Crystals of nitrate of urea.

but in others these appearances have been absent, the spasm and loss of power being due to the effect of the altered blood on the muscular tissue.

*Examination of the urine.*—The urine is to be examined as soon as possible after it has been obtained from the animal, as it quickly becomes ammoniacal. In every case its specific gravity is much increased—1·185 or higher. It is highest during the first few hours after attack, as it often becomes lighter in colour, even in those cases which succumb after the first day or two. Boiled, the urine generally gives no reaction, except that ammoniacal gas is emitted. Tested with nitric acid, it occasionally becomes almost solid, but the solidity is rarely due to coagulation of albumen, but to the precipitation of crystals of nitrate of urea in great abundance. These crystals appear as mica-like scales of a brown colour, and fall to the

bottom of the glass. At first the addition of the acid causes much effervescence, the urine being strongly alkaline, containing ammonia carbonate. When the effervescence has ceased, the urea is rapidly precipitated, and the liquid loses much of its dark colour. Microscopically examined with a quarter-inch objective, it presents the appearances seen in fig. 56.

In some samples of urine analysed by Ivison Macadam sugar has been detected.

If the serum of the blood is carefully prepared, the crystals of nitrate of urea can be detected in it by the following process:—

Take a given quantity of serum, and precipitate the albumen by boiling, filter and evaporate the liquid to dryness over a water bath. Treat the residue with alcohol, which dissolves urea readily, evaporate the alcoholic extract to dryness, and add a little water, so as to make a syrupy mass, which should be plunged into a freezing mixture, and a few drops of pure nitric acid added to it, when crystals of nitrate of urea will soon be found in it.

There is no doubt that this malady attacks mares more particularly during the period of oestrus. This can be accounted for in two ways, independently of any association with hysteria, namely—(1.) Because mares whilst in this state are very often kept off work for a few days; and (2.) They are in a highly excitable nervous condition, and more apt to suffer from spasmodic diseases, the causes of which may be very trivial.

In the spring of 1870 I was called to see a horse in a dying state from this disease. I found that four had already died from it. They were farm horses, which in consequence of the frost had not been worked or exercised for some time. As long as they were kept in the stable they seemed all right, and immediately on the frost giving way they were put to work. They were all attacked, and succumbed in a few days; some of them dying early in the disease, others living for some days; and, what was very remarkable in those which struggled longest, the muscles lost all power of contractility, and in the one I saw there was seemingly no *rigor mortis* after death.

*Treatment.*—The therapeutics of this disease may be summed up in a few words, namely,—keep the various excretory organs acting

freely, in order to assist nature in expelling the degraded products from the circulation. For this purpose a cathartic or oleaginous aperient ought to be administered without delay. The kidneys generally act freely enough, and need no stimulus; but should they cease to perform their functions, diuretics, and more particularly colchicum, are to be used. The animal should always be placed in a large, well-bedded, dry, loose box, with plenty of short straw or chaff around it, which is to be removed as soon as it becomes damp or wet. If unable to rise, it is to be turned from side to side two or three times a day, or more frequently if it becomes uneasy. Enemas are to be administered until purgation commences, and plenty of diluents allowed, and the urine must be frequently removed from the bladder by means of a catheter. Generally the horse is thirsty, and will drink cold water freely and to manifest advantage.

There is no necessity to aggravate the disease by either stimulants or sedatives. They always do harm during the first stages of the malady. If, however, towards the third day the animal seems depressed, moderate doses of spirits of nitrous ether may be given two or three times daily; and about this time attempts should be made to get the horse on its legs. If unable to do so without assistance, the slings should be placed under it, and it is to be gradually placed on its feet. At first it will be much disinclined to stand, and will be apt to fall from muscular weakness; but if the limbs are supported and smartly hand rubbed for some minutes, they will regain much of their lost power, the horse will begin to "feel himself," as is commonly said, and will often commence to feed and do well. It should be kept in the slings for a few days, but taken out occasionally for a little exercise; and when allowed to lie down for the first time, it should be carefully watched, as it is apt to knock about very much if unable to rise with ease.

During the first few days the diet is to be of the lightest description and restricted in quantity, but when convalescence has commenced, it must be moderately nutritive and easy of digestion, as much muscular debility exists in the digestive apparatus as well as in the voluntary muscles. At this stage a few doses of *nux vomica* may be given with advantage.

I have never seen any benefit accrue from external applica-

tions to the loins or back. On the contrary, fomentations, frictions, stimulants, or blisters increase the irritability of the animal, cause it to struggle when it otherwise would remain quiet, and do much harm.

If the pathology of the disease be borne in mind, the practitioner will at once see that the symptoms will cease as the morbid material in the blood is eliminated, and that the first and great aim of treatment is to effect this, after which the muscular debility must command his attention; and if there are indications of failure of the heart's action in two or three days after attack, stimulants are to be administered with freedom.

Any of the ordinary stimulants will answer the purpose, but perhaps we should exclude ammonia, as the blood-poisoning products are too similar to that medicament.

#### OXALURIA.

A condition of the system manifested by dulness, capricious appetite, loss of flesh, debility, stiffness about the loins, a bran-like scurf on the surface of the body—pityriasis—"hide-bound," and the presence of the oxalates of lime in the urine.

This disease is most commonly seen in hunters or other horses whose work is irregular, and which have to undergo long periods of fasting, as during a long and severe day with the hounds, the result of such long fasting being a weakened or debilitated condition of the digestive organs, whereby the products of the primary digestion are imperfectly formed, and unfitted for assimilation and consumption in the processes of nutrition and calorification.

The disease is also induced by food rich in saccharine matters, as carrots, turnips, or other roots, more especially if the digestion is deranged. Oxalates may also appear in the urine during recovery from other diseases, more especially during a protracted recovery from an enzootic or inflammatory affection.

Oxalic acid is a product of imperfect combustion or oxidation of the amyloid and nitrogenous matters of the body. It may also result from an imperfect condition of those matters primarily; a perverted acid condition of the digestive organs, impediment to the respiratory function, or from a superabundance of food, more especially if combined with insufficient

exercise. Various vegetables contain oxalic acid—the sorrels—but I am not aware that these are partaken of by the horse, or that they are ever the cause of this disease. The pathological importance of a deposit of the oxalates has been considered doubtful by writers on human medicine. Some writers affirm that they result from chemical changes in the urine whilst in the renal passages, and that their occasional presence is no indication of disease. In our patients, however, their continual presence generally attends a form of indigestion in which an acid condition of the alimentary tract is a constant accompaniment.

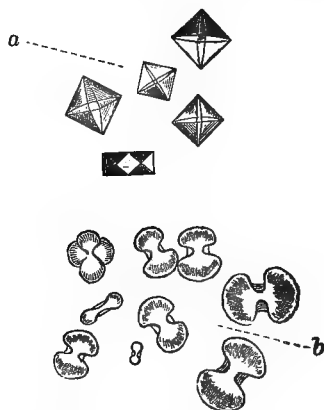


FIG. 57.—Crystals of oxalate of lime.  
(a.) Octahedral form. (b.) Dumb-bell form.

*Semiology.*—Loss of flesh ; capricious appetite ; a dry, immovable, scurfy condition of the skin ; the horse is said to be hide-bound and scurfy ; a desire to lick the walls, and frequent urination of a palish amber-coloured urine, the act of micturition being often accompanied and followed by signs of uneasiness, such as whisking of the tail, or even attempts to lie down, or kicking at the belly with the hind feet. The mouth has sometimes an acid or sour smell, the tongue is covered with a whitish fur, and there is generally a degree of constipation or irregular action of the bowels. An animal thus affected is stiff in the loins, and is easily fatigued.

*Etiology.*—The causes are to be found in irregularity of feeding, want of sufficient exercise, irregular exercise and work,



such as fatiguing journeys with long intervals of rest; improper food; or it may originate in weakened digestion from an unknown cause.

*Condition of the urine.*—The urine at each emission is scanty; of a pale amber colour; clear, like pale sherry, and is neutral or acid in its reaction. If examined immediately with the microscope, crystals of the oxalates will most probably be detected. In some instances, however, the crystals cannot be detected with the microscope for a day or two after the urine has been passed, the oxalic acid being present in its free state, and afterwards combining with the lime and forming at least two distinctive forms of crystals:—1st. and most common, Octahedra (*a*); 2d. Dumb-bell (*b*) (the dumb-bell is very rare in the horse).—See fig. 57.

These crystals are soluble in nitric acid without effervescence, are insoluble in water, and are unaltered by boiling in acetic acid or liquor potassæ.

Beneke, a German pathologist, has studied the question of the origin of oxalic acid in the human frame, and has arrived at the following conclusions:—

“1. Oxaluria, a condition which accompanies the lighter or severer forms of illness, has its proximate cause in an impeded metamorphosis—that is, an insufficient activity of that stage of oxidization which changes oxalic acid into carbonic acid.

“2. Oxalic acid has, if not its sole, its chief source in the nitrogenous constituents of the blood and food: everything, therefore, which retards the metamorphosis of these constituents occasions oxaluria.

“3. Such a retardation of the metamorphosis of the azotized constituents of the blood may be determined by the following causes:—

“(a.) Abuse of azotized articles of food (direct retardation).

“(b.) Abuse of saccharine and starchy articles of food (indirect retardation).

“(c.) Insufficiency of the red corpuscles, and (eventually) diminished oxidation.

“(d.) Insufficient enjoyment of pure, fresh, ventilated air.

“(e.) Organic lesion, which may in any way impede respiration and the circulation of the blood.

“(f.) Conditions of the nervous system which bear a character of depression, whether these arise primarily from mental derangement or from pathological states of the blood.

“4. Excess of alkaline bases in the blood, which, as numerous observations tend to show, plays an important part among the etiological conditions of oxaluria; and it is not improbable that an increased production of lactic and butyric acids in the digestive canal, consequent thereupon, impedes the development of the red corpuscles, and thereby generates that chlorotic state which so often occasions and accompanies oxaluria.

“5. Catarrhal conditions of the intestinal mucous membrane, in case they are accompanied by oxaluria, have at most only a common source. They may determine oxaluria by causing deranged digestion, but cannot be considered as its proximate cause.”

Oxalic acid differs from carbonic acid in possessing an atom less of oxygen and one less of carbon, and we cannot be surprised at its occurrence in the urine, when we remember how slight a deviation in the oxidation of tissue may cause its formation.

*Treatment.*—In the first instance, it is necessary to alter the deranged condition of the digestive apparatus, and for this purpose a purgative is often rendered imperative. When the bowels have been operated upon, the condition of the system (the oxalic acid diathesis) is to be overcome, and the digestion improved by mineral acid tonics, the nitro-muriatic acid in preference to others, given in a decoction of gentian or other vegetable stomachic bitter. The diet must be carefully regulated, and all roots and food containing much sugar withdrawn. The quality of the drinking water must also be looked to, and if it contains an excess of lime, it must at once be changed, for if the oxalic acid is to be eliminated from the system it must be removed in a soluble form, and every remedy will fail if an excess of lime is present in the circulation. For the same reason alkalies generally are not to be administered, as they are transformed into oxalates, more or less insoluble, in the body.

Bearing in mind that non-oxidation is a source of oxalic acid, the practitioner will order regular exercise, pure air, and good grooming.

The irritation manifested in the passage of the urine is due to the direct irritation of the oxalic acid and oxalates on the mucous membrane of the urinary passages, and the frequency of the act of micturition is due to the same source. If not very harassing to the animal, no special treatment is required for its removal, for as the urine regains its natural character the irritation passes away. Should it be really a source of much annoyance, enemata containing belladonna or opium may be used.

In some cases of oxaluria, when the mineral acids have failed, the disease has rapidly disappeared when acid phosphate of soda has been substituted.

#### ASTHMA (BROKEN WIND).

Asthma, like diabetes, might with propriety be classified amongst constitutional diseases, for now and then it arises from no cognisable cause. I have, however, deemed it advisable to classify it with the dietetic diseases, as in the great majority of instances it originates from errors in feeding.

*Definition.*—A non-inflammatory disease, characterised by difficult and peculiar breathing; the inspiratory movement is performed with ease, the expiratory by two apparent efforts. The difficulty in breathing is constant, but is liable to remissions and severe exacerbations. A peculiar cough, called “the broken-winded cough,” is a constant symptom; indigestion and flatulence aggravate the dyspnoea.

*Etiology.*—The causes of broken wind, as laid down by writers, are very numerous and complicated. Youatt says that emphysema of the lungs is present in almost every case that he has examined. Haycock ascribed the disease to hepatization and induration of a portion of one or both lungs, chronic disease of the digestive organs, or of the nerves of the respiratory apparatus, rupture of the diaphragm, and inflammation of a low subacute character of the mucous membrane of the bronchial tubes and minute air cells. Gibson attributed it to enlargement of the contents of the chest. Dr. Lower supposed that it arose from rupture of the diaphragmatic nerve. The Professors of the Veterinary College at Lyons (1826), after performing a variety of experiments, were led to conclude that the dyspnoea arose from a reversed situation of the diaphragm, caused by nervous derange-

ment. M. Godine, Jun., professor at Alfort, considered it as a natural defect in the normal and relative proportions of the right and left side of the heart. M. Demussy, in a Memoir to the Royal Society of Agriculture of Paris (1823), affirms that by direct observation he discovered that those districts of France where hay or other dry food is most used, are subject in a peculiar degree to broken wind among their horses.

Coleman said it originated in mechanical rupture of the air cells. Blaine supposed it arose from emphysema of the lungs, due to the formation of gases within the lung tissue.

To give my own opinion, I have no hesitation in asserting that broken wind is generally due to improper food, more particularly to bad, musty, or coarse hay, containing a large quantity of woody fibre, from being allowed to become too ripe before being cut, and to a superabundant allowance of hay of any kind, with a deficient supply of corn.

Mr. Anderson, V.S., Glasgow, has very carefully investigated the causes of broken wind, and has been led to conclude that in most instances it is due to habitual over-loading of the stomach with coarse, indigestible food; in some cases the stomach has been found much enlarged, and containing masses of dry food adherent to its walls.

In some districts the disease prevails, or used to prevail, to a great extent. In some parts of Wales I have very frequently noticed many broken-winded horses; and upon inquiry, have discovered that their fodder has been of the worst possible description. I quite agree with Professor Gamgee that broken wind is at first a purely nervous affection, dependent on the condition of the digestive system, and in which the pneumogastric nerve is especially involved, and that the organic lesions are the effects of the nervous disorder. Some horses are peculiarly prone to suffer from broken wind, where no fault can be detected in the method of feeding. Such animals are constitutionally predisposed to indigestion, tympany, and irregularity of the bowels; they are generally round, shallow-chested horses, but are not bad thrivers before they become affected in the wind.

*Pathology.*—The smaller air tubes are surrounded by involuntary muscular tissues; in broken wind, owing to the irritation of the par vagum, the function of this muscle is inter-

fered with. In the early stages, and during the paroxysms of broken wind, it is spasmodically contracted; in the later stages most probably it is paralyzed; in either condition there is arrest of respiration. The irritation arises from the action of indigestible food upon the cardiac branches of the vagus, and is reflected to the pulmonary branches of the same nerves. Recent experiments by Bert have convinced him that the lungs contract under the influence of the vagus, branches of which, it will be remembered, supply both the lungs and stomach, as well as other organs. The action of the bronchial muscle surrounding the tubes is brought into play during the expiratory movement, whereby the lungs are assisted in expelling the *tidal air*. The same thing happens in the lungs of a horse suffering from broken wind as in the lungs of a human being suffering from asthma. Owing to nervous irritation, there is arrest of respiration, and this can occur either in expiration or in inspiration; the arrest during expiration being the more easily induced, indeed "in some animals it is impossible to obtain arrest during inspiration."—(PAUL BERT.) The arrest takes place in the act (inspiratory or expiratory) which happens to be going on at the instant of application of an irritant to the nerve; "the lung being, as I take it, seized and fixed by spasm, and immobility of the bronchial muscles."—(Dr. J. C. THOROWGOOD.)

In the horse the arrest is always during the expiratory act, which, in consequence, becomes prolonged, difficult, and calls for a double contraction of the abdominal muscles to force the air out of the distended and now immobile bronchial tubes. I have noticed that very round-chested horses sometimes become broken-winded without apparent cause, and I conclude that difficult expiration may occur in them from limited thoracic contraction. When the conformation of the chest is round, respiratory movements, at least thoracic movements, are very limited in extent; for when the chest is naturally round, its conformation is but little altered during the respiratory movements. An animal so formed is incapable of taking a deep inspiration, and as incapable of performing a strong expiration; the lungs of such contain much residual air, the thoracic walls are more resonant on percussion, and in them probably arrest of expiration is most easily induced. Every horseman knows that round-chested horses, although apt to put on fat and look well, are not so

capable of performing the same severe and fast work as deep-chested animals; in fact that they are "not so good in their wind."

The abnormal condition of the bronchial tubes and air vesicles is at first due to purely nervous derangement, but this is very rapidly succeeded by organic or structural change. Inflation of the air cells to excess prevents the free circulation of the capillary blood-vessels, and thus the nutrition of the bronchial muscle and mucous membrane is weakened; degeneration succeeds, and the lungs become more and more emphysematous; the air cells undergo dilatation, their parietes become stretched and disorganized, and air now infiltrates into the meshes of the connective tissue of the lungs. In this manner both vesicular and interlobular emphysema may be induced. Various other changes may occur in the thoracic organs, the most constant being hypertrophy and dilatation of the right side of the heart. By bearing these facts in view, much of what seems irreconcilable may be understood, and what have been looked upon as causes will be seen to be but mere effects.

*The morbid anatomy* of broken wind differs according to its duration at the time of the animal's death, which is generally due to another disease or to an accident. If death occurs soon after asthma has been manifested, no morbid change may be detected. Professor Dick said he had examined many cases where no disease could be discovered. In other cases the stomach has been impacted with food. This impaction of the stomach will only be observable in an animal which has died suddenly, or has been killed, as in one suffering from another disease the appetite would be impaired, and the horse would not eat for a time prior to its death.

If the disease has continued for a period of time, the following lesions may be detected:—Emphysema of the lungs, chronic thickening of the bronchial tubes, dilatation of the air vesicles, with anæmia, degeneration, and rupture of their walls, pallor of the pulmonary parenchyma, and eccentric hypertrophy of the right side of the heart, or the whole cardiac substance may be variously altered in structure.

*Symptoms.*—The inspiratory movement is performed with ease, but the expiratory by two apparent efforts, at the conclusion of which the muscles relax, and the flanks fall with peculiar force. There is a singular cough, which seems to be "ejaculated with a

sort of grunt through the upper part of the trachea, perhaps from a sympathetic connection with the parts below."—(BLAINE.) Auscultation will detect the respiratory murmur weakened or absent; there is a loud, sonorous, sibilant wheeze, heard particularly loud towards the posterior part of the chest. Rhonchus and sibilus of every tone and variety may be heard all over the chest. There is stagnation of air in the lungs, and the sounds may change both in character and site, owing to the varying clonic contractions and relaxations of the bronchial muscle. A rubbing sound is heard, if rupture of air cells has occurred, denoting the presence of air in the lung tissue. On percussion, the resonance of the thoracic walls is increased, showing that the lungs are distended with air; the chest itself seems rounder than is natural, and the thoracic movements are very limited, whilst the abdominal ones are violently increased during a paroxysm. The symptoms are much more severe after the animal has been fed, and after the spasmodic fit has passed away, the breathing may be tolerably easy until it is fed again. The digestive organs are weak and easily disordered; flatulence is a prominent symptom; and the animal is often harsh and dry in its coat, pot-bellied, and unthrifty.

*Treatment.*—Great attention to diet. The food and water should be carefully regulated as to quantity, and be of the best quality. Feeding on bran mashes, containing a few ounces of linseed oil with lime water, has proved serviceable in the practice of Mr. Anderson of Glasgow. A purgative should occasionally be administered, and the alimentary track always kept in proper order.

The old farriers used to make a supplementary anus for the horse by passing a red-hot iron into the rectum from immediately below the tail, through which a leaden ring was inserted, to keep it patent. There is no need to resort to this barbarous and insane method; but they supplied a reason for doing it, namely, that the artificial anus allowed the flatus to escape from the bowel easily, and that it did away with the disagreeable sound which accompanies that act when it is naturally performed.

The symptoms of broken wind may be palliated by all methods which improve the digestion, and by remedies that are calculated to give them tone; hence arsenic, alkalies, bitters, more especially nux vomica, and the various tonics are serviceable. All

mere remedies, however, must be of secondary importance to hygiene and dietetics.

"Horse-coupers" resort to various methods for relieving the breathing of broken-winded horses. These persons know well enough that the animal breathes moderately well when the stomach is empty; they therefore take good care to keep it short both of food and water, and give it a sharp trot to unload the bowels. Shot, lard, gunpowder, opium, and other remedies are then poured down its unoffending throat, and most of these remedies seem to exercise a sedative or "stilling" effect, and the unwary purchaser only knows too late how cleverly he himself has been "sold."

There is one consolation, if not for the buyer, at least for the public generally, and that is, broken wind, like many other diseases the results of ignorance, is fast becoming a thing of the past.

#### LEAD POISONING—LEAD PALSY—PLUMBISM.

*Definition.*—A series of morbid phenomena induced by the absorption of the salts of lead contained in solution in the drinking water or in the food.

Lead poisoning is a disease generally confined to those districts of the country where lead smelting is carried on. It may, however, occur accidentally in any part where animals have access to lead-paint, or splinters of leaden bullets, which are often scattered about in quantities near rifle targets. It also occurs in cattle pastured in fields manured with town ashes, which often contain scrapings of paint-pots, waste paint, &c.

In whatever form the lead gains access to the stomach, it must be rendered soluble before it is absorbed into the circulation. Lead-paint, which is splashed on grass when ring-fences or railings are being painted, or the scrapings of painters' pots thrown on the pasture, leaden bullets, and other insoluble forms of lead, are acted upon by the acid secretion of the true digestive stomach, and are thus rendered soluble and fit for absorption into the animal economy. The portions of insoluble lead found in the rumen, &c., of horned cattle are to be looked upon as indicating that lead is present, and that some of it is absorbed into the system, rather than



as actual causes of any symptoms present prior to death. Lead in its metallic state appears to be devoid of medicinal or poisonous action. Four ounces of shot were given to a dog at the Veterinary School of Lyons without effect. The metal may sometimes, however, be converted within the body into an oxide or active salt.—(FINLAY DUN.) Shot is used by low horse-dealers to relieve the symptoms of broken wind, and doubtless some of the lead is dissolved by the gastric acids, and acts as a sedative upon the gastric nerves.

Plumbism, or lead poisoning, results from the absorption of lead into the circulation. It is thus diffused through the whole body, and exerts its toxic action upon all the organs and tissues of the animal frame, but it does not affect all parts alike, but accumulates in some more than in others.

Dr. George Wilson (see *Monthly Journal of Medical Science*, May, 1852) found that the spleen yielded lead most abundantly; next to the spleen the liver yielded most; then the lungs, afterwards the kidneys, then the heart, and the intestines least. Lead has also been found in the brain and in the muscles; and the blue line observed in the gums of animals and men poisoned with lead is supposed by Toures to be due to the lead in combination with the tartar of the teeth.

Lead having once entered the body, leaves it very slowly, and may be detected in it months after an animal has ceased to receive lead with its food or water. It enters into combination with the tissues of the body, and is retained in the system as an insoluble compound, and only leaves the body on the breaking up or disintegration, medicinally or otherwise, of such insoluble compound.

The symptoms of slow lead poisoning in the lower animals are, capriciousness of the appetite, loss of flesh, convulsive fits, a blue line seen on the gums where teeth are present, the gums in the interdental spaces showing no trace of this colour. Colic is sometimes absent, and constipation is not so constant a symptom as in man; indeed I have repeatedly seen a diarrhoeic condition of the bowels with leaden-coloured and foetid stools. In the horse, roaring is considered, in Wales, the first symptom of lead poisoning, and every roarer in the lead districts about Holywell and Flint is said to have had lead (*plwm*). Mr. Shenton of Bakewell, in a letter to Mr. Finlay Dun, thus de-

scribes the symptoms of lead poisoning in eleven horses which came under his observation in 1852:—"There was a rough, staring coat, a tucked-up appearance of the abdomen, and a slightly accelerated pulse; in fact, symptoms of febrile excitement, which usually passed away in about a week. About this time large quantities of grey-coloured matter were discharged from the nostrils, and saliva from the mouth; but at no time was there enlargement of the submaxillary, lymphatic, or salivary glands. Neither was there constipation of the bowels, which appears to be nearly always present in cases of lead poisoning in man. Fits and partial paralysis came on at intervals; and when the animals got down they often struggled for a long time ineffectually to get up again. The breathing up till this period was pretty tranquil, but now became so difficult and laboured that the patient appeared in danger of suffocation. The pulse was in no case above sixty or seventy; and I ascribed the difficulty of respiration to a paralyzed state of the respiratory apparatus. The animals did not live more than two or three days after these symptoms appeared.

"The *post mortem* appearances varied but little. The lungs and trachea were inflamed; the lungs engorged with large quantities of black blood; the trachea and bronchi filled with frothy spume. In all cases but two the villous part of the stomach presented isolated patches of increased vascular action; and in all cases the intestines, especially the large ones, were inflamed. The blind pouch of the cæcum was nearly gangrenous. There was nothing remarkable about the liver, spleen, or kidneys, except that they were of a singularly blue appearance. The brain and spinal cord were not examined."—(FINLAY DUN'S *Materia Medica*.)

In the diagnosis of lead palsy in man the electric current plays an important part, for it is found that when the palsy is induced by lead the excitability of the muscles is much diminished, whilst it is normal in ordinary paralysis. "*Therefore, when the muscles of a paralytic limb move well under the influence of the electric current, we may fairly conclude that there is no lead in the system.*"—(Dr. ALTHANS on *Paralysis, Neuralgia, &c.*)

Mr. Herapath reports in the *Chemist* for 1855 (see F. Dun's *Materia Medica*) some interesting cases of lead poisoning, which followed the erection of smelting furnaces on the Mendip Hills

in 1853. The inquiry appeared to commence half a mile from the chimney, and to extend half a mile further. Oxide, carbonate, and sulphate of lead were found on the herbage, hedges, and hay. On the live stock "the effects of the metal were, a stunted growth, a leanness, shortness of breathing, paralysis of the extremities, particularly the hinder ones, the flexor muscles of the fore legs were affected, so that they stood upon their toes, swelling of the knees, but no constipation or colic as in the human species; in a few months death followed. In the young the symptoms were more conspicuous and the mortality greater. Lambs were yeaned paralytic. When three weeks old they could not stand, although they made great efforts to do so. In attempting to feed them from a bottle they were nearly suffocated from paralysis of the glottis. Twenty-one died early out of twenty-three. Colts also died; and those that lived could not be trotted 150 yards without distressed breathing. Pigs confined to the sty were not injured, but if allowed to roam were soon affected. The milk of cows and sheep was reduced in quality and quantity, and cheese made from the former had less fat in it. I found in the milk of both minute traces of lead. The dead subjects showed the mucous surfaces to be paler than natural. The lungs had large portions of a dark red colour, with circumscribed edges, not like ordinary inflammation, but evidently surcharged with fluid. There was a blue line seen in the gum of the lower jaw, which Dr. Taylor said in court was not caused by lead poison, as it did not occur, as in the human subject, on the upper edge of the gum, but where the gums first come into contact with the teeth, about three-sixteenths of an inch below the top edge. I therefore dissected out this line, which was about three-quarters of an inch in length, and the thickness of sewing cotton; and by aid of carbonate of soda and the blowpipe reduced a spangle of lead from it, quite visible to the jury without the aid of the microscope."

I have seen several valuable hunters poisoned by water conveyed in leaden pipes, and the cause of the solution of the lead pipes arose from contamination of the water at its source with decaying vegetable matter. In all these cases the animals became roarers, and each time they drank of the poisoned water their teeth became black.

*Treatment.*—In the rapidly developed form of lead poisoning

from the accidental ingestion of lead paint, bullet spray, &c., the treatment must be directed to prevent the solution of the lead in the stomach and intestinal canal. Firstly, in the ox and sheep perform rumenotomy and remove the contents of the paunch; then, by administering such agents as enter into combination with lead, form insoluble combinations. For this purpose the bowels are to be acted upon by a solution of sulphate of magnesia, to which sulphur and sulphuric acid have been added. By this means the lead in the intestines is converted into an insoluble sulphate and sulphide. If pain be present, opium or hyoscyamus is to be added to the purgative. The acid is to be continued for some time, as it is only by keeping the digestive apparatus charged with it that the solution of the lead by the gastric juices can be prevented, for the thin crust of sulphate, which probably forms on the surface of the portions of lead, may be thrown off, and the metal, or its oxide or carbonate, as the case may be, again exposed to the action of the gastric juices.

In addition to the purgative and acid, diluents are to be freely administered, and purgation actively kept up for some days. It is also necessary to keep up a copious discharge of urine, with the view of eliminating the absorbed lead from the body.

When the bowels are thoroughly acted upon, and if the sulphuric acid does not seem to agree with the animal, sulphur and vegetable tonics may be substituted. The sulphur is partially transformed into sulphuretted hydrogen, which combines with the lead, and forms an insoluble sulphide.

In the slower form of lead poisoning, and where the lead has been slowly accumulating in the system, it will be well to consider the chances of success before attempting a cure; and in none but the mildest cases should this be attempted, as the termination will prove neither creditable to the practitioner nor profitable to the owner.

In all cases a small dose of the sulphate of magnesia is to be administered, and when this has operated, the elimination of the poison is to be attempted by the iodide of potassium.

The principle upon which the iodide of potassium acts has been pointed out by Melseus, who assumes that the lead is retained in the body as an insoluble compound with the tissues.

The iodide, after its absorption into the blood, combines with the lead, and forms with it a new and soluble salt. The poison is thus liberated from its union with the injured part, dissolved out from the damaged fibre, and once more set afloat in the circulation, and along with the remedy cast out with the urine. Great caution is at first necessary in using this remedy, as at the moment when the compounds, fixed in the body, become dissolved or transformed, the phenomena of acute poisoning may occur. The dose must therefore be small at first, and either increased or diminished, as the patient seems to bear it.

Galvanism is also recommended for lead poisoning in man; and, if necessary, may be tried in the case of a valuable horse or cow.

It will be apparent that removal of animals from the source whence the lead is obtained must be the first step in the treatment.

The soluble salts of lead, such as the acetate, when given in poisonous doses, cause nausea, quick, small, hard pulse, colic, delirium, or staggers, stiffness of the limbs, paralysis of the optic nerves, insensibility, and death; and on *post mortem* examination the gastric mucous membrane is found reddened from congestion, or grey, and having a macerated appearance, owing to the chemical action of the salt.

#### PARAFFIN-OIL POISONING.

I have met with several instances where cattle, horses, and sheep were slowly poisoned by drinking from a stream into which the refuse from paraffin works was discharged, or by eating grass and hay which had deposited upon it some of the oils which are given off in the distillation of paraffin shale.

The symptoms were, gradual loss of flesh, and a persistent diarrhoea, and it was only on a *post mortem* examination being made that the cause of death was discovered. The appearances were as follows:—

Anæmia, dropsy, and anasarca, wasting of the muscles of the body generally; absence of fat, and other signs of a prolonged malnutrition; the characteristic lesions were discovered on opening the abdominal cavity. The intestines had a greyish-

black appearance throughout, and were covered with very dark grey or blackish spots of a dull appearance. These spots were found to consist of the various intestinal glands, surcharged with the pigmentary matters of crude paraffin. The glands of the mesentery were more or less enlarged, and presented the same dark appearance; the lacteals were of the same colour and were found to contain paraffin; the liver presented nothing very unusual, but the kidneys, more especially on their inferior surfaces, were covered with the dark spots.

The animals in one case which I had the opportunity of seeing, had been removed from the field, through which the polluted stream flowed, some months prior; they had continued to eat moderately well, but everything they took seemed to pass through the intestinal canal as if the power of absorption had been entirely lost; and when the condition of the glands and lacteals was examined, this could be accounted for; indeed, it may be said that the animals had died from slow starvation, arising from impermeability of the chyle vessels, due to their being blocked up by crude paraffin. Although so long a time had elapsed since the paraffin had obtained access into the body, the characteristic smell was retained; and even many months after they were removed from the body and preserved in spirits, the odour was still recognisable. Several actions at law have been tried, and in most cases the owner of the injured stock was successful in proving damage.

#### RHODODENDRON POISONING.

Several cases of poisoning by the *Rhododendron hybridum* having been observed by my late student, Mr. Gunn, Veterinary Surgeon, Beaulieu, he kindly wrote me the following particulars, which are well worthy of attention:—

“On 8th April last I was asked to attend a lot of fourteen cattle, one and two-year-olds, and on examination the following symptoms were noticeable:—staggering, swaying from side to side, partial paralysis of hind extremities; some of the animals would press their heads to the wall and strike out with their fore limbs, as if endeavouring to get forward, then vomit a green ropy spume. One, a yearling stirk, was unable to get up, the hind extremities being completely paralyzed; eyes staring and fixed

in their sockets, pupils not dilated, mucous membranes very pale. great straining, passing small quantities of hard, dark foetid faeces covered with mucous. The symptoms varied very much, as some of the animals, especially the two-year-olds, would lie down during the spasms of pain, and kick the belly with the hind legs. Vomition was a marked symptom, and came on at intervals, when the animal would groan with pain, vomit, then a spasmodic cough came on, which lasted for a few minutes. When administering medicine, if the animal's head was much raised above the level of the body, the creature would instantly fall as if struck with an axe. On examining the substance vomited, it was full of the flower buds, leaves, and young shoots of the rhododendron in a semi-masticated state. An inspection of the park grazed by the cattle was made, when about three cart-loads of the prunings of rhododendron were found thrown over the fence by the gardener; the prunings having been recently, as was seen, eaten of by the cattle.

“*Treatment.*—Linseed oil, with potass carbonate, and small doses of tincture opii in the cases suffering from colic was in every instance successful. The yearling stirk affected with paralysis was treated as above, with the addition of the iod. potass., which in a few days had the desired effect.

“*P.S.*—The first symptoms of illness were noticed fifteen hours after the cattle had eaten part of the prunings.”

#### YEW POISONING (TAXUS BACCATA).

Considerable difference of opinion seems to exist as to the poisonous or non-poisonous properties of the leaves of the yew tree; some maintaining they are not poisonous except in a partly dried or decayed state, whilst others state that cattle can eat them with impunity, either dry or green.

Numerous instances are, however, recorded which prove that the yew is poisonous to cattle, sheep, pheasants, &c.; and my own experience enables me to agree with this opinion, namely, that the yew, English and Irish, is poisonous, both in its green and dry state.

The symptoms are rarely noticed, as the toxic action is very rapidly fatal, destroying life without there being any very decided manifestation of symptoms beyond loss of appetite,

great prostration, feeble pulse, coldness of the surface of the body, and rapid sinking of the vital powers.

The *post mortem* appearances are equally unsatisfactory, and but little can be detected beyond the presence of the leaves and twigs in the stomach, and indications of a narcotico-acrid poison, and sometimes a great congestion of the brain or even an apoplexy.

In investigating into the cause of death of one lot of cattle poisoned, several of which were found dead, it was discovered that some clippings from yew trees had been thrown into the park the previous evening, and in those which had died first the poison was found in the greatest abundance; others of the cattle lingered for several hours, and in these the leaves were less in quantity.

From what I observed in making the examination, it would appear that it is not essential that the yew leaves should pass beyond the rumen to exert their toxic properties, as in some of the cases no leaves were found beyond the viscus, whilst in others a few were found in the reticulum; in those that lived the longest some of the leaves had reached the omasum. No doubt some of the juice expressed during mastication would become absorbed and cause death.

*Treatment.*—The indications for treatment are the removal of the toxic vegetable as quickly as possible; and, provided the cause has been discovered by the history of the occurrence, or the *post mortem* examination of the animal or animals already dead, and seeing that vomition is induced with so much difficulty, I see no objection to the performance of rumenotomy, in order to remove all the contents of the rumen; but before resorting to such an operation, the practitioner should endeavour to overcome the effects of the poison by large and repeated doses of stimulants, such as ammonia and alcohol, friction to the skin, and warm clothing. Should this succeed, the operation may not be necessary. At the same time, it must be remembered that, even if the first effects be thus obviated, the animal will continue in danger until the poison has been expelled. The process of rumination re-established will again subject the plant to the action of the saliva; its remaining toxic properties being thus set free may induce a fatal collapse, while perhaps all has been thought secure. Purgatives will be essential, and ought to be administered without delay.



CAKE POISONING—CASTOR OIL SEED (*RICINUS COMMUNIS*) AND  
CROTON SEED (*CROTON TIGLIUM*) POISONING.

It is well known that both castor oil seed and croton seed are very poisonous, even a less quantity than one per cent., when present in the food—usually feeding cakes—causing serious illness, and even death. Mr. J. W. Leather, in *The Analyst*, July 1892, says: “It requires but a very small quantity of these seeds—probably three or four seeds or less per lb.—to cause serious illness or death to stock.” Mr. Leather was kind enough to communicate his method of detecting this adulteration of feeding cakes to me in a letter, June 12, 1892, at which time I was engaged in investigating several outbreaks of a disease of a serious and fatal nature occurring amongst cattle fed on a particular linseed cake. His method I found to answer every purpose, and I have since repeatedly tested it. Boil about a pound of the material with one to two per cent. of hydrochloric acid for one hour. Decant the upper liquid from the sediment several times; boil the residue with dilute alkali for another hour, then wash by decantation until the washing water is colourless. Treat the residue with half a pound to a pound of bleaching powder (chloride of lime), and add sufficient water (a quart to three pints) to keep the temperature from rising. This is frequently stirred and then left to stand for several hours, although the bleaching action is generally complete in about four hours. It is then repeatedly washed by decantation, and all the lime removed. This process renders the testa of the seeds generally met with in cattle foods sufficiently transparent to be recognised by the microscope,—in fact bleached, some being completely decolorised; whereas in the case of the croton and castor oil seeds the outer integument is washed away, and the principal part of the testa remains black and opaque, and at the edges some indication of the structure can be seen.

Subjected to the action of sodium hypochlorite, even for several days, the castor and croton seeds retain their colour, whilst those of linseed, cotton, locust bean, &c. are readily bleached. Free chlorine gas has a more powerful action, and will gradually bleach both croton and castor seeds, but it requires more time, and even at the end of three or four days their colour is not entirely removed.

After passing through this bleaching process the castor-oil seed testa has a much smoother and shiny exterior than the croton seed, and this is an important point in their distinction, as microscopically the structure of each is very similar, being built up of closely packed bundles of fibres of the same thickness; but on the outermost edge of a transverse section of the croton testa there is thickening of each bundle of fibres, absent in a section of castor oil seed.

Experimentally Mr. Leather has added the testa of castor oil seed, weighing 0·223 gramme to 1 lb. cotton cake, subjected it to the bleaching process, and has recovered 0·200 gramme; and out of 0·074 gramme croton seed testa mixed with 1 lb. cotton cake recovered by picking out 0·060 gramme. In performing these experiments it was found that a mouldy croton bean became bleached after the lapse of a day. When microscopically examined, this bean was found to have been penetrated through and through with a fungus. It may be mentioned that the hilum of cotton seed does not bleach so readily as the other portions, but there is no difficulty in distinguishing it, for the pieces are round, have no sharp edges, and are marked by alternating rings of grey and black on the surface. If the material to be acted upon is roughly handled, the testa is broken into small fragments, and may thus be overlooked.

*Differences in the appearance of the outer integuments of the Castor and Croton Seed.*—*Castor Oil Seed before bleaching.*—The seeds are oval, and bluntly rounded at one end, while at the other they come to a point, where there is an aril, easily broken off. The testa or outer covering is smooth and glazy, grey in colour, with very characteristic longitudinal striations of dark-brown, yellow-brown, or black.

*Croton seeds* are oval and triangular or imperfectly quadrangular in shape; but, as in the case of castor seeds, are only seen in the ground state, in cakes. The testa or outer seed coat is cinnamon-brown in colour, and when scraped shows a blackish appearance, and underneath the brittle testa there is an inner pale-coloured part which surrounds the yellow endosperm.

*Symptoms.*—Both croton and castor oil seeds have similar effects upon the animal economy. The seeds having had the fixed oil expressed from them, still contain an active poison in

the embryo, which is included in the cake. Five and a half ounces of castor seeds will cause death in a horse. In other animals relief is obtained by the poison acting as an emetic and cathartic. In a short but variable time, depending upon the extent of the adulteration, the animals fed upon such cakes present symptoms of great prostration, uneasiness, and excessive diarrhœa, the watery alvine evacuations containing clots of blood and large quantities of mucus. In calves stomatitis is a prominent symptom, the mouth being covered with thick layers of epithelium, which readily peel off, exposing the mucous membrane red and congested in appearance; there is much flow of saliva, and champing of the lips, as seen in foot and mouth disease.

The *post-mortem* appearances are those of gastro-enteritis.

*Treatment.*—Demulcents, such as starch, gruel, linseed tea, with opium and chalk, to allay irritation, and some antiseptic, such as carbolic acid or small doses of soda hyposulphite, to arrest stercoral putrefaction, with stimulants to overcome the great depression and prevent a fatal collapse. Eggs, milk, and other fluid nutrients may also be necessary in order to overcome the feebleness of the heart's action.

Powerful astringents should not be administered in the earlier stages, as they, by checking the outpouring of fluid from the vessels, prevent the removal of the poison and increase the congestion.

#### LATHYRIASIS.

A disease induced in the horse by feeding on a leguminous seed called the *Lathyrus sativa*,—the vetchling. Professor Stewart Macdougall, of the New Veterinary College, has given an interesting account of this plant in the *Veterinary Journal*, January 1895.

He says the natural order Leguminosæ includes a very large number of plants, having among them diverse properties, some being nutritious, others purgative and astringent, and still others poisonous. The lathyrus belongs to the sub-order Papilionaceæ, which also contains lupins, whin, broom-clover, vetches, haricot bean, scarlet runner, peas, gram, lentil, &c.

There are several varieties of *Lathyrus sativa*, showing slight differences in colour of flower, in size and colour of seeds, in

colour of foliage (darker or lighter), and in strength of growth; but they have one characteristic in common, namely, in being wedged, angular (triangular), or hatchet shaped. They differ in colour in various countries where they are grown. According to Harz, quoted by Macdougall, the variations are as follows:—

Locality.	Colour.	Largest Diameter.	Weight of 100 seeds in Grammes.
Carinthia, . .	Greenish yellow, . . . .	10·1	20·871
Carinthia, . .	Reddish yellow, . . . .	10·3	25·830
Austria, . . .	Yellowish green, . . . .	9·5	23·756
Sicily, . . . .	Pea-coloured, spotted, . .	12·0	41·423
Bohemia, . . .	Reddish pea-coloured, . .	8·3	16·637
Verona, . . . .	Pea-coloured, . . . . .	15·0	56·545
Greece, . . . .	Partly faint pea, partly brown,	7·6	18·438
Sicily, . . . .	Pale pea, . . . . .	14·7	48·991
East India, . .	Dark marbled on brownish ground, . . . . .	7·1	13·619
East India, . .	Deep reddish brown, black spotted, . . . . .	7·5	16·527

The nutrient ratio is 1:1·75, the characteristic being richness in nitrogenous constituents. The seed contains 84 per cent. of dry substance, yielding 25 per cent. proteid, 1·9 per cent. fat, 54·5 per cent. non-nitrogenous extract, 4·1 per cent. fibre, and 2·9 per cent. ash. For further information upon the botanical and other characters of the plant the reader is again referred to Professor Macdougall's article.

So far as my knowledge goes, only two kinds of the seeds have been concerned in the production of the disease, namely, a small dark-coloured seed, known as Indian Mutters, brought from India mostly as ballast to such ports as Liverpool and Glasgow, and a larger white variety, popularly called the dog-tooth vetch or dog-tooth pea, and sometimes sold under the name of Russian peas.

The terms Matar, Muther, Mutter are used in India for peas generally. Whether the seeds used are of the smaller dark kind or of the larger white kind, they have been equally injurious.

Professor Macdougall says that he found the seeds in poultry

food in several of the Edinburgh grain-dealers' shops, added as an adulteration. Some of the dealers did not trouble about the presence of the lathyrus seeds, whilst others objected to them, not on the ground that, so far as they knew, the seeds were injurious, but that they were an inferior quality of pea.

Dr. Voelcker states that he has discovered several cases where feeding cakes have been adulterated with lathyrus, and that injurious effects followed their use.

The effect on man using the seeds as daily articles of food is a sudden and incurable paralysis of the lower limbs, and there are records of epidemics of paralysis following their use, both in Asia and Europe.

*Symptoms in the Horse.*—The animals, seemingly in a perfect state of health whilst at rest, on being put to work or even exercised, particularly if the weather be cold, are seized with roaring and great difficulty of breathing, some immediately dying from asphyxia unless tracheotomy be performed, others gradually overcoming the dyspnoea and remaining apparently well whilst kept in repose.

The following is the description of the symptoms given by the manager of the Bristol Tramway Company,—case at law reported.

123 horses out of a stud of 800 fell ill owing to feeding on mutters. "The horses suffered from what was styled, for want of a better name, 'an epidemic of falling.' They would fall suddenly, without any accountable reason. Cab and carriage shafts were broken daily, as also were the car poles." And again, "A horse being exercised was taken suddenly ill; it roared, its flanks heaved, its mouth was kept wide open, the nostrils were distended (dilated), and the tongue hung out and became livid; it stared and staggered, and threatened every moment to fall down strangulated and suffocated, and during this paroxysm, which lasted several minutes, the perspiration ran freely off every part of the horse."

Principal M'Call, who was consulted in the Bristol case, having seen much of the disease in Glasgow, and who advised tracheotomy, with the best results, says that mere excitement is sufficient to induce a paroxysm.

In 1884 the Messrs. Leather, veterinary surgeons, Liverpool, discovered the disease in a stud of 74 cart horses, out of which

35 became ill, 19 died, 2 were killed as useless, and 14 recovered. They had 3 or 4 lbs. of mutters mixed with their ordinary food per day, which consisted of 20 lbs. of grain, for three months. Professor M'Call states that 2 lbs. per day could be continued for six weeks or so before symptoms of the attack become apparent; and it is remarkable that upon some of the horses in these studs, as well as in others that have come under my observation, the poisonous qualities of the seeds seem to have no ill effects.

In Liverpool, Glasgow, and Bristol it appears that the dark seeds only were used, but there are several reports from Bedlington,—a number of pit ponies; Eastwood, 20 or 30 horses; Newcastle, 12 or 15; near Sheffield, reported by Mr. Abson, V.S.; 12 horses at Hamden Colliery, near Birmingham,—where the disease was induced by the large light-coloured seed.

Both in man and the horse the lathyrus is a cumulative poison, the symptoms showing themselves the sooner the greater the proportion of seeds contained in the food. It appears from the evidence of many observers that boiling renders the seeds innocuous. Thus the late Mr. Thomas Greaves told me that he knew that they had been used regularly for years after being boiled without any bad effect; and Professor M'Call mentions that in the neighbourhood of Glasgow 100 bolls of the seeds lay unused for some time, until they were by his orders boiled, steamed into a pulp, and given in quantities of  $1\frac{1}{2}$  lbs. per night per horse, without any bad effect. In the case of human beings Watt says "there is a certain capriciousness" in the effects of the poison on different individuals; and, adopting the view that the poisonous effects are due to a volatile alkaloid, he suggests that persons eating thin cakes made from the sativus grain do not suffer, as all parts of the cake being exposed to a high temperature, the poison is eliminated, whereas in food preparations exposed only to moderate temperature sufficient of the poison remains to act injuriously on those partaking of such food.

Professor Macdougall states that the seeds cause paralysis and spasm in pigs, and that when fattened on the meal they lost the use of their limbs, but became very fat whilst lying on the ground. Cattle seem to resist this poisonous affect, but it has been stated that the seeds are injurious to milch cows, and that sheep fed on small quantities receive no harm. Pigeons are said to lose the power of flight. Geese eat them with impunity. Poultry do not eat them readily.

The true nature of the poison is not satisfactorily determined, but may be a volatile alkaloid. Astier claims to have isolated an alkaloid. Further experience has convinced me that the organism found in an extravasate form around the throat of one of the Liverpool horses was an accidental complication, as extravasation was not witnessed in any except the Liverpool horses. The mutters in that instance were very dirty, and fouled with rodent excrement, from which a microbe was cultivated homologous in appearance with that found in the extravasate.

The North of England Institute of Mining Engineers, at a meeting held at Newcastle-on-Tyne, December 14, 1895, formulated the opinion "that a reasonable quantity of these peas is eaten with impunity by pit horses in that district." The "reasonable quantity" referred to turns out to be, according to the statement of Mr. Hunting, a quarter of a pound per day, and from what is known I have no doubt but this is a correct statement. If this could be correctly carried out, doubtless it is probable that no bad effects would follow. To my mind, however, there is a risk of imperfect mixing, which would result in a greater share of the seeds being eaten by one horse than by another.

At the same meeting Mr. Clement Stephenson stated that Mr. W. Hunter, M.R.C.V.S., Newcastle-upon-Tyne, had discovered that all the affected horses, upon which tracheotomy had been performed, had recovered. Mr. Hunter, however, stated that they had not entirely recovered, but had greatly improved in their breathing, and worked daily without distress, although the tubes had been removed some months ago.

#### LUPINOSIS.

A disease said to be due to the ingestion of plants belonging to the genus *Lupinus* of the Natural Order *Leguminosæ*.

The whole subject of Lathyrism appears to be very little understood. In India during famine periods the natives eat these peas, and there are some cases of paralysis resulting. It would appear to be difficult to induce these symptoms by experimental feeding of the seed, and it is possible that there are other matters for consideration than the pea itself being

the cause. Experimental research on the subject is necessary.

The name lupin is said to be derived from *lupus*, a wolf, because the plants "devour all the fertility of the soil," but the derivation is doubtful. None of the lupins, numerous though the species are, and very common in our gardens, are natives of Britain, their chief home being the countries in the neighbourhood of the Mediterranean Sea and the temperate regions of North and South America. The members of the genus are shrubs or sub-shrubs, and can be recognised by their digitate leaves. One of the best known is the species with white flowers—*Lupinus albus*—grown in Southern Europe, either for fodder or as a manure for the purpose of ploughing the crop in. The Greeks and Romans, in spite of the bitter taste of the seeds, acquired a liking for them, and used them as an article of diet. Cattle, too, are known to have been fed on them after the bitter taste had been removed by steeping and boiling. The yellow lupin (*Lupinus luteus*) has sometimes been recommended as a forage plant in places where a poor, light, and sandy soil would grow nothing else. In spite of its bitterness, which made it unpalatable to stock generally, sheep are said not to refuse it, and to thrive on it. One authority remarks that cases of poisoning have been known to follow its use, but were not understood. Others speak of lupin seed meal as excellent for young calves.

A bitter principle, probably alkaloidal, seems to be characteristic of the various species. We have certain knowledge in the case of two at any rate, viz., *Lupinus albus* and *Lupinus angustifolius*. From the seeds of these an alkaloid has been isolated, called lupanine. Bitter to the taste, it is said not to be hurtful to man, but experimentally to be poisonous to frogs.

*The Root Tubercles of Leguminosæ.*—The Leguminosæ are well termed "nitrogen collectors," for they collect quantities of nitrogen, and this without making the soil poorer, but, on the contrary, leaving it richer in nitrogen at the end of their growth. Experimentally it is known that in the absence of nitrogen in the soil they can flourish.

The Leguminosæ, then, possess some means of supplying themselves with nitrogen, means denied to the generality of plants, which are dependent almost entirely for their nitrogen on the



nitrates in the soil. The Leguminosæ, almost alone among plants, are able to make use of free nitrogen of the air or soil, and this power is associated with the presence of nodules or swellings in their roots. These swellings vary in size in the different species. Examination by the microscope of a transverse section through such a swelling shows a multiplication in number of the cells of the ground tissue of the root. In these cells there can be seen, in addition to the protoplasm, a number of bacteria. These bacteria pass into a form to which the name "bacteroid" has been given. These bacteroids expose a large surface to the air, and make use of the free nitrogen preparatory to its being combined into such forms as the plants can absorb and use. We have here a useful partnership between a lower plant and a higher, between bacteria on the one hand and the leguminous plant—bean, clover, lupin, &c.—on the other. The bacteria are first of all nourished by the plant, and later the contents of the bacteroids are absorbed by the plant.

If a soil be sterilised and the germs thus killed, no tubercles will be formed until the soil be in some way "microbe seeded."

If this disease were due to a ptomaine or toxine generated by the action of the bacilli upon the constituents of the plant, the fact that lupinosis is only induced in animals grazed on poor pastures could be easily understood, but conditions identical with those observed in lupinosis are seen in this country, and the disease prevails as an epizootic in certain parts of North America, particularly in two counties of Nova Scotia and Antigonish, where it is called "The Pictou Disease;" and M'Eachran of Montreal, who investigated it in 1880, came to the conclusion that it was not due to the ragwort, the plant supposed to be the cause by the inhabitants, but to the deficiency of nutritive materials in the animals' food,—a conclusion supported by my experience—see Cirrhosis of the Liver—and also by the observations of Continental farmers, who believe it due to the exhaustion of the soil, weakened for want of proper cultivation; and it is remarkable to observe that lupin when first grown upon poor land may induce the disease, but is innocuous from land in which it has been grown for a number of years.

Again, in Nova Scotia the farmers are also fishermen, and allow their farms to become deteriorated for want of proper cultivation.

There is nothing said in the report of the "Pictou Cattle Disease" as to whether lupins are grown in the particular districts where the disease prevails, but they are plentiful in America. I am therefore in the dark on this point, and am forced to suppose that it is probable that a toxine may be formed in other leguminous plants when grown in poor soils.

The disease is said to attack the horse, ox, goat, sheep, and deer; inoculable in the dog, but the rabbit seems to have immunity.

The disease seems to be most prevalent amongst sheep in Prussia, Silesia, Hanover, North Germany, committing great havoc; and in the bulletin of the Royal College of Economy of Prussia, February 1880, it is stated that out of 240,000 sheep in one district of Pomerania, 14,000 and 13,000 died in one year. In the Nova Scotia report there is no mention of sheep.

The disease seems to appear in two forms, viz., an acute form, characterised by acute hepatitis, the liver soft and friable, and in some instances enlarged by fatty infiltration, the fat supposed to be transported from the subcutaneous adipose tissue to the liver; this is followed, if the animal live, by an acute hepatic atrophy.

Dr. Wyatt Johnston, Montreal, says—"The condition of the liver in the early stages is a very peculiar one. There is a very great secretion of bile; the gall bladder is very much distended with bile, sometimes containing over a quart. Notwithstanding this, there is an excess of bile passed in the stools, which are dark, and contain large quantities of bile.

"In the early stages the liver shows marked signs of parenchymatous degeneration, or cloudy swelling; later on a fatty change in the liver cells is very common.

"In the later stages of the disease,—that is to say, in animals which have been ill two or three months, the liver always shows a marked condition of cirrhosis, and the liver cells have disappeared, and are replaced by delicate fibrous tissue. . . . There is a peculiar condition of the submucosa of the fourth stomach, which is distended with a clear viscid fluid, so that the folds are swollen out in round dropsical masses, the distention being due to the fluid. In some cases, when the cattle have been ill for over a month, a number of ulcers are seen in the stomach. In the sheep the changes are similar to the above;

the alterations of the liver consist mainly in chronic interstitial hepatitis, with hypertrophy of the connective tissue (see Cirrhosis of the Liver) congestion of the portal system, followed by ascites, serous tumefaction of the spleen, and of the gastrointestinal mucous membrane."

*Symptoms.*—In the Pictou disease, the symptoms, as described by M'Eachran, are loss of condition, with diarrhoea at an early stage, and the milk acquires a bitter taste and unpleasant odour. This change in the milk is often the earliest symptom; there is a soft pulse; a very prominent bulging of the eyes; a roughness of the coat. In about a week the abdomen begins to swell owing to the development of ascites, and if the animal is killed in from one to three weeks from the commencement of the symptoms, the ascites is abundant, the fluid amounting to several bucketsful. With this ascites there is a disappearance of the fat about the kidneys, so that the adipose tissues become filled with a clear fluid, which apparently replaces the fat. In some cases the temperature remains normal, although the pulse is increased in rapidity.

As described by Continental writers, the symptoms in sheep are loss of appetite and elevation of temperature, which falls considerably before death: this elevation is sometimes observed on the day following the ingestion of toxic lupin. Symptoms of jaundice generally, but not always, appear on the second, third, fifth, or sixth day, and may come on suddenly, or slowly and gradually, being first seen in the eye; there is also weakness and stupefaction, stiffness of gait, the recumbent posture being generally maintained; in some cases great excitement, grinding of the teeth, spasmodic agitation of the jaws, and in some instances true trismus from the third to the twenty-second day after the attack. Constipation may be an early symptom, the faeces coated with yellowish or pitchy-brown mucus mixed with blood. Diarrhoea follows. Chronic lupinosis, said to be induced by a prolonged use of moderately toxic plants, assumes the form of anæmia without the symptoms of jaundice.

In the horse, induced by lupin seeds in the oats or litter, the symptoms are those of gastro-enteritis, cerebral depression, grinding of the teeth, head carried low, with uncertain and staggering gait, elevation of temperature—106° F. in some cases—accelerated respiration, more or less marked jaundice, constipa-

tion, the fæces being covered with mucus and having a foetid odour, diminished secretion but frequent emission of small quantities of highly coloured urine. These symptoms continue for about a week, and recovery is complete in about sixteen to twenty-one days. Directions are given for the treatment of lupinosis, which must be mainly preventive, there being no specific antidote. When impossible to exclude the lupin, it is recommended that it should be mixed with good food and given preferably to old animals; that the toxic fodder should be put in small heaps and left exposed to the action of rain, which washes the poison from the superficial layers, or to macerate the toxic lupins for forty-eight hours in a solution of soda—1 in 100—renewing the liquid from time to time, or to subject the lupins to the action of steam for at least two hours under the pressure of at least two atmospheres.

When the disease has manifested itself, it is recommended that alkaline liquids should be avoided, as they dissolve the toxine and increase the danger, and that some acid should be added to the water in order to render the lupin and toxine insoluble, and the poison removed from the intestinal canal by oleaginous purgatives.

I have endeavoured to condense much of what has been written on lupinosis for the benefit of students particularly, but I have very grave doubts as to disease being due to the lupins, for not only have we similar conditions in the Pictou disease, but in this country acute and chronic diseases of the liver are frequently met with, particularly in sheep—see Liver Diseases—and on the Continent. Hautner has seen identical conditions in sheep fed on malted potatoes, and several observers state the same conditions are induced by inundated pastures and by pea, bean, and vetch stubble, &c.

From Gamgee's work on Toxicology the following notes have been obtained on hemlock, water hemlock, water parsnip, meadow saffron, and monkshood:

#### HEMLOCK.

HEMLOCK LEAVES, HEMLOCK ROOT, from *Conium maculatum*, but several plants are popularly included under the name Hemlock.

The extract of the leaves and roots of the common or spot hemlock has been found by Dr. Christison to produce paralysis of the voluntary muscles, with occasional slight convulsions; then paralysis of the respiratory muscles; and lastly, death from apnœa, the heart continuing to contract long after respiration had ceased. Opposed to these results are the observations of Mr. Judd, who found that in cats, doses even not large enough to be poisonous, caused great languor and drowsiness, and often profound sleep for two or three hours, the muscle excitability being lessened, and the circulation and general temperature reduced. After death, the appearances are general venous congestion, fluid state of blood, and softening of brain.

In the *Veterinarian* for 1845, Mr. Read has spoken of lambs poisoned by eating the *Conium maculatum*. They became giddy, listless, could not move about, struggled and died. On opening the rumen, Mr. Read was struck with the beautiful green colour and peculiar odour of the contents. The hemlock thickly covered a boggy spot in the field where a number of sheep were feeding. Only three died; the rest were saved by bleeding, and a solution of sulphate of magnesia, acidulated with sulphuric acid, was given to each.

Mr. Holford, of Northwich, was at one time called to attend a stock of dairy cows that had been turned out the previous day on a marshy piece of ground surrounded by a brook. The latter must have overflowed during heavy rain; the ground was covered with coarse grass of all kinds, and much of the common hemlock. On entering the cow-house, of those that appeared to be worst, Mr. Holford found three seemingly lifeless; but, on close examination, he with difficulty found the pulse, of an extremely feeble character, beating; but little more than half so rapidly as when in health. The extremities were deathly cold, but respiration not much altered. The eyes were closed; and on elevating the lids the pupils were much dilated, and the retina unaffected by light. All the animals were comatose, the bowels inactive, the skin cold, and hair standing erect. These were the symptoms of three; but, during the day, twenty-one more of the stock exhibited similar appearances in a milder form. One cow that was bled by the owner died; the others were kept up by

stimulants every few hours, evidently rallying after each dose, and if the medicine was discontinued, they sank into a state of lethargy. During the hours of the night stimulants were administered eight times, and perspiration at length broke out on the skin, the extremities became warm, the countenance lively, the bowels responded, and in a week or two the animals regained their original strength. Lecocq saved a cow by bleeding, and giving 12 ounces of acetate of ammonia in water, in two doses. Noll saw two goats poisoned by common hemlock. Ducks have been seen stupefied and paralytic from eating the seeds of the plant. Milk and oil saved seven out of nine affected by the poison.

The treatment of poisoning by hemlock consists in emptying the rumen of cattle and sheep, and inducing vomiting in the dog. In all animals the strength must be supported by stimulants, and artificial respiration resorted to should the breathing have ceased.

*Analysis.*—Hemlock yields a volatile oil upon distillation with water, which appears destitute of noxious properties. The active principle is a peculiar volatile alkaloid, conia, which exists in the plant, combined with an acid—probably the conic—by which it becomes fixed, so that it is not given over with water in distillation. Conia is a yellowish liquid of oleaginous aspect, strong, penetrating mouse-like odour, and very acrid, benumbing taste. The mouse-like odour can very readily be perceived when the leaves of hemlock are triturated with a solution of caustic potash in a mortar, and affords a valuable test. Conia is an energetic poison, its effects being identical with those of hemlock itself. It is obtained by distilling the plant with caustic potash.

THE WATER HEMLOCK (*Cicuta virosa*) is often left untouched on pasture ground by horses and cattle. In Sweden and Norway, goats and sheep—but especially the latter—are affected by the poison. Donkeys become vertiginous from it. It is said that in Norway, pigs eat it without danger, and birds likewise are unaffected by partaking of the seeds. Weidmann observed a two-year-old ox suffering from the effects of the cicuta. The symptoms were loss of appetite, distended belly, hanging and feeble gait, pulse quick and irregular. Blood-letting seemed to do harm;  $\frac{1}{2}$  ounce of

muriatic acid in 3 quarts of water appeared to be more beneficial.

Damitz saw four head of cattle die four hours after eating the fresh root. Seven others were taken with tetanic spasms and other symptoms, which disappeared by bleeding and the administration of strong vinegar. Frause speaks of horses dying from eating hay which contained water hemlock. The head and neck were bent to the right; the look was dull, eyes strongly withdrawn in the orbits, pupils dilated; involuntary mastication; bluish-coloured mucous membranes; pulse indistinct, heart-beats 120 to 130, and respirations 26 to 30 in the minute. Death occurred with convulsions.

ENANTHE CROCAT (the hemlock water dropwort or water parsnip) is considered one of the most virulent of English vegetable poisons. M. Bellamy, veterinary surgeon at Rennes, published some experiments on this poison in the *Recueil de Médecine Vétérinaire*. The symptoms noticed were accelerated breathing, foaming at the mouth, injected mucous membranes, pulse feeble and frequent, tetanic spasms or violent convulsions of the limbs, symptoms of colic, and, lastly, paralysis of the hind extremities and death. The canine species would seem to be less affected by the poison than man, horse, or cattle. Bellamy recommends in the treatment of cases of poisoning by this agent, to use blood-letting, emetics, and purgatives, mucilaginous draughts, and cold applications to the head.

#### COLCHICUM AUTUMNALE.

Professor Weiss has been at some trouble to collect information regarding poisoning by meadow saffron, which is the most common of plants proving injurious to domestic animals. The latter will generally avoid it, unless pressed by hunger, or if it be cut up and mixed with other substances. Mr. Musgrave says that cattle will graze amidst the deep green tufts of meadow saffron, seldom touching it, except they come from a distance or from a different pasture, when they do not seem to recognise its nature at once, and partake of it freely, until its poisonous effects, characterised by profuse, liquid, muddy, and foetid stools, are produced. The leaves, stems, and seeds are all noxious, but especially the seeds, which, if

swallowed, are said to adhere to the coats of the stomach, and induce, at the several points of adhesion, spots of inflammation, which occasion death. Colchicum acts on the stomach and intestines as an irritant, on the brain as a narcotic, and favours decomposition of the blood.

Death sometimes occurs rapidly from paralysis, but generally animals survive several days. Hübner mentions an instance of three cows dying an hour after partaking of meadow saffron in the green state. Horses and oxen that survive remain for weeks dull, and have an awkward gait.

Stolz and others observed pigs, after eating of the unripe seed capsules, to have the visible mucous membranes a blue colour, to be dull, nipped in at the flanks, weak gait, tail depressed, eyes dull and closed, cold extremities, foaming at the mouth, vomiting of green frothy matter, diarrhoea, with foetid and blood-stained fæces. Most pigs retain their appetite and desire for drink. Frey observed difficult breathing and accelerated pulse, shrunken abdomen, and liquid, greenish evacuations per anum. According to Stolz, twenty out of thirty-two pigs, from two to four months old, died presenting the above symptoms. Three out of four pigs died from partaking of a boiled mess containing colchicum and potatoes.

The *post mortem* appearance consisted in distension of the stomach, with inflammation and gangrene of it and of the intestines; blood dark and thick; the brain congested. Milk and animal charcoal were exhibited as antidotes.

Gierer, Trachsler, Aschmann, Hierholzer, and others have spoken of cases of poisoning in the horse by colchicum. The animals were dull, without thirst or appetite; in some cases the thirst was excessive. The mucous membranes were of a dark blue colour; eyes dull, expression staring, pupils dilated; pulse quick, scarcely perceptible; abdomen distended and urine dark; occasionally there were no fever symptoms. In the cases that were not severe the breathing was scarcely disturbed; but in others it was difficult, panting, and accompanied with sighs and groans. Hierholzer observed colicky pains, rolling, sitting on the haunches, no discharge of fæces or urine. After death, inflammation and gangrene of the mucous membranes of the stomach and intestines were observed; the lungs were gorged with blood. In the treat-



ment of these cases Aschmann recommends marsh-mallow decoction with fœnugreek seeds, with elder and chamomile flower decoction; the animals to be warmly clothed and warm fomentations to be applied to the abdomen. The horses that were spared remained weak, and with a feeble gait for fourteen days. Mr. Morton has spoken of the sedative, laxative, diuretic, and diaphoretic properties of colchicum given in 3-ounce doses twice daily. The animals evinced no disinclination for food, but it was rather thought that the appetite had increased.

When cattle partake of large doses of meadow saffron, according to Musgrave, the first effects consist in purging of liquid fæces, which are muddy and very offensive; extreme depression of the general system, and especially of the circulatory apparatus, the pulse being irregular and scarcely perceptible, and the exhalants on the surface of the body called into augmented action from apparent debility. Mr. Musgrave first observed the poisoning effects of meadow saffron, when three bullocks became quickly purged after eating the leaves and seed-vessels of the plant, which had been separated from the new hay, that the latter might not be too much heated. On another occasion Mr. Musgrave was requested to see eight yearling heifer calves which had just been brought from Gloucestershire, and two days previously turned into a pasture where the meadow saffron grew in considerable abundance. It was at the time in full leaf, and beginning to seed. Mr. Musgrave found all the calves more or less affected, and presenting the following symptoms: Violent purgation, the dejections being liquid, of the colour of muddy water, and extremely fœtid. Two were lying down and unable to rise; the rest stood with their backs arched, their coats staring, the abdomen of some of them swollen, and they all seemed afraid to move. Their ears and heads were drooping; the eyes were glassy, and the pupils dilated; the nose dry and cold, as were the ears and other extremities; the pulse was irregular, and too quick and weak to be counted. On the two lying down, perspiration stood in drops on the hair like dew. The urine was small in quantity and dark in colour. Five out of the eight died. Kufener has observed no discharge of excrement, and Frey speaks of diminution in the

quantity of milk secreted. Lindenberg has seen many of the above symptoms, with staring coat, grinding of teeth, evacuation of blood with the fæces, and twitchings of the limbs, with slow and difficult movements in progression.

The *post mortem* appearances are, according to Lindenberg, those of carbuncular fever, with the veins gorged with blood; the alimentary canal the seat of extravasation of blood, but not of true inflammation. The first and second stomachs contained liquid matters, whereas the contents of the third and fourth presented nothing particular. The bladder contained red urine. Musgrave mentions peritoneal inflammation and inflammation of the omasum.

“The treatment of cases of this kind must always depend,” says Mr. Musgrave, “on the severity of the symptoms when the animal is first seen. Under all circumstances, the beast should be at once housed or clothed; then, if the poisoning be only in the first stage—that is, before the circulation is much depressed or very violent purging has set in—it would be as well, perhaps, to give a strong oleaginous purgative at once, with the twofold view of overcoming the action of the poison and forcing it onwards, a practice which seems to be very successful with the farmers I have alluded to. But if, on the contrary, it should have arrived at a more advanced stage (and it is rare that it will not be so when the practitioner is called upon to attend), when the general system shows considerable depression and violent purgation, with liquid fœtid stools, have commenced, the first object must undoubtedly be to allay the pain and irritation set up, by giving a full dose of opium and camphor, combined with linseed-oil; also, apply some strong stimulating embrocation to the belly and extremities. If the symptoms still continue severe, throw up emollient injections with opium; drench freely with linseed-tea and gruel, repeating the opium if found necessary, and even adding ether, according to the state of depression, until the unfavourable symptoms begin to subside. Should any signs of debility remain, a dose or two of a carminative tonic and astringent will seldom fail to effect all that is required to perfect the cure. But if these measures prove of no avail, nothing that I am aware of will save the animal. One circumstance connected with these cases is worthy of special

remark—namely, that in every *post mortem* examination I have made, oak buds and leaves have been found with the colchicum amongst the ingesta. They looked fresh, as if just eaten, and were scarcely discoloured. It struck me that the animals might have been instinctively led to eat these astringent substances as a remedy for their sufferings; and this view was strongly confirmed on turning out some affected animals, when they began immediately to browse on the oak-boughs within their reach, and would touch nothing else. Animals after their recovery may be safely turned again into the colchicum meadows, as they will ever after refuse the plant.”

MONKSHOOD (ACONITUM NAPELLUS).

This plant, indigenous in the European Alps and common in gardens, is a most virulent poison. Its flowers are large and violet-coloured. The roots somewhat resemble a cluster of radishes, and have been mistaken for horse-radish, with fatal consequences. All animals are poisoned by aconite; but cases of accidental poisoning are chiefly observed amongst the young cattle in Switzerland. It has been said that goats on the Austrian mountains eat the plant without suffering. Hertwig has seen sheep, and still oftener goats, eat the flowers of monkshood, and die speedily afterwards. Hübner says that goats that eat of it become affected with tympanites, vomit frightfully, and have a staring and anxious look. They recover without treatment; and the milk secretion, which is checked from the influence of the poison, soon returns. The symptoms produced in the horse by aconite are coma, efforts to vomit, cold sweats, difficult breathing, small and thready pulse, and paralysis of the hind-quarters.

Dr. Fleming considers that death by aconite may occur (1) by producing a powerful sedative impression on the nervous system; (2) by paralyzing the muscles of respiration, and causing apnoea; and (3) by producing syncope.

*Analysis.*—Aconite owes its active properties to an alkaloid called aconitina. The taste of aconite is at first bitter, but after a few minutes gives rise to numbness and tingling of the lips, is believed by Dr. Christison to be sufficient to distinguish the poison from others. When the plant has been

the cause of death, the contents of the stomach should be carefully examined, with a view to detect any fragments of it. Besides noticing the botanical character of them, small portions should be chewed in order to observe the highly characteristic tingling which the aconite plant produces. An alcoholic extract of the organic mixture may be made and examined (1) by tasting it; (2) by applying a small quantity to the eye, the extract of aconite produces contraction of the pupil. Instead of merely making an alcoholic extract, the contents of the stomach may be subjected to Stas's process. The product obtained by it, although not possessing the physical characters of aconitina, will exhibit its physiological action in a very marked degree, if a considerable quantity of the plant has been taken.

#### ACONITINA.

The most formidable poison yet discovered. It is an alkaloid contained in all parts of the aconite, but especially the root. Its chemical reactions are not characteristic, and we must therefore rely upon its physiological action when searching for it.

#### JAVA BEANS (PHASEOLUS LUNATUS).

In the wild state these beans are purple and very poisonous; in cultivation the colour becomes modified to a yellowish tint, with stripes or blotches of a violet colour.

In this cultivated state the beans are not so poisonous, and after prolonged cultivation may become quite innocuous.

These beans contain certain elements—phaseolunatin and emulsion—which, when the beans are heated and moistened, unite, and, among other things, form hydrocyanic acid.

A quantitative analysis of the Java bean of commerce revealed the fact that each pound of Java bean-meal was found to contain *nine maximum* doses of prussic acid for an adult.

The statement has been made by exporters of Java beans that the latter become safe to use after being boiled in water, and Messrs. Tatlock and Thomson have stated that when Java beans are steeped in water and afterwards boiled, a

considerable proportion of the prussic acid-yielding glucoside is removed. Experiments made at the Imperial Institute with Java beans have shown, however, that practically no change in the quantity of glucoside present is effected by this means, but as the activity of the enzyme is destroyed, the ground boiled beans no longer liberate prussic acid when mixed with water.

There is on record one case which seems to indicate that this treatment of Java beans is insufficient as a precaution against poisoning. Thus Robertson and Wynne state (*Zeit. Anal. Chim.*, 1905, xliv. 735) that four persons out of seven who had made a meal of *cooked* "kratok" beans (kratok is a vernacular name for Java beans in use in Holland and Germany), died, and in each case clear proof of poisoning by prussic acid was obtained.

Within a few minutes after the ingestion of the beans or the bean-meal there are sudden toxic symptoms, and almost immediately the animal dies.

Professor McCall, of Glasgow Veterinary College, carried out some experiments with samples of the bean-meal used in one case. The material was given to a collie dog, a short-horn cow, and a black and white cob, with the result that they all died, the dog within two hours and the cow and horse within one hour of the first appearance of the symptoms.

A noticeable feature in the experiments was the period of time which elapsed—namely, fifteen or twenty minutes—between the ingestion of the Java meal and the appearance of toxic symptoms. The explanation is that the poison does not exist in the form of free hydrocyanic acid in the beans; but the elements are there, and on the addition of moisture and heat (both of which are furnished by the stomach) a chemical combination occurs, resulting in the slow and gradual production and liberation of the deadly poison.

When these conditions are produced artificially outside the body, the formation of the poison goes on gradually for several days.

#### COMMON BOX (*BUXUS SEMPERVIRENS*).

Its leaves contain a bitter, acrid poison. A pound and a half will cause acute enteritis and death in a horse.

## POTATO POISONING

occasionally occurs in horses which either get a surfeit of potatoes at the beginning of the potato season, when they are unaccustomed to them, and in these cases the symptoms are those of great bowel irritation, diarrhoea, and death; but in other cases, when the animal eats unripened potatoes, the solanin acts as a poison, and induces symptoms similar to those as described by Bissange in bovines and pigs, as follows: He observed poisoning in those animals in nearly 800 cases, due to the exhibition of potatoes. The cases are common in certain years by reason of the peculiar climatic conditions that favour changes in the tubers. The changes are various; the potatoes may be frozen, sprouted, green, or spoiled; the parings and the tops may also prove hurtful. Whatever condition sets up the disease, the symptoms of poisoning are sensibly the same. At the onset there is loss of appetite, absence of rumination, muscular tremors, tottering gait, weakness of the hind-quarters. Later there is violent diarrhoea, the dejections have a peculiar and strong odour, there is tympany in the left flank, sometimes ptyalism, and always pupillary dilatation, producing a peculiar appearance of the animal, and which readily lends itself to detecting the affected animals in the stable. There is somnolence, and, it may be, also vertigo. The conditions last three or four days; the loss involved is serious, having regard to the loss of condition that follows, to the falling off of the milk supply, and to the abortions which sometimes occur. In the pig there are both vomition and diarrhoea, but recovery is more rapid.

*Treatment*, as followed by Bissange, consists in the administration of tannin in doses of from 15 to 30 grammes daily, or of similar drugs. The *tannin* appears to neutralise the solanine, which is not yet absorbed. Stimulants are indicated to ward off depression, and aromatics (coffee) were given and external stimulants applied. The author has made some experiments with solanine, and his conclusions are as under:

1. Uncooked potatoes, even when sound, although they may produce an increase in the amount of milk secreted, ought to be given to milking cows in small quantities only;

they irritate the digestive tract and often determine a gastro-enteritis.

2. Cooking brings out the nutritive qualities of the potato; it removes acidity, and partially destroys the harmful action of solanine.

3. Frozen and diseased potatoes ought not to be utilised for feeding, because of the solanine contained. Moreover, they are poor in nourishment; the chief food principles have disappeared.

4. Sprouted potatoes should not be used until the sproutings have been removed from the tubers.

5. If the potatoes have become green, they should only be utilised in small quantities, and then only after they are cooked.

6. Potato tops should only be used for litter, and should not be used for feeding purposes.—*Bulletin Vétérinaire, ex Annales.*

#### DOG MERCURY (MERCURIALIS PERENNIS).

Horses and cattle sometimes eat this plant, and as a result suffer from nephritis and enteritis.

Mr. Secker Smith, M.R.C.V.S., Barnsley, forwarded me the following history of two cases he recently met with—subjects, a three-year-old cart colt and a yearling cart filly:

“The colt, who was quite well in the morning, after showing signs of great abdominal pain and passing bloody urine, succumbed before I saw him.

“The filly was in good health the previous day, and on examination I found her to be breathing heavily; to have her back arched; pulse 79, and weak; temperature 104·3°; visible mucous membranes of a deep yellow colour, and there was a continuous dribbling of urine, mixed with blood and with what appeared to be decomposing blood.

“I made a *post mortem* examination of the colt, and found the stomach, bowels, and peritoneum all deeply stained yellow. The stomach was full of food, and in it I detected portions of *dog mercury*. The kidneys were inflamed; the pelves filled with coagulated blood. Other organs were healthy, except the liver, which was much congested.”

## DARNEL GRASS.

Speaking of grasses in general, Lindley says : "None are unwholesome in their natural state, with the exception of *Lolium temulentum*—darnel grass—a common weed in many parts of England, the effects of which are undoubtedly deleterious, although perhaps exaggerated of *Bromus purgans* and *catharticus*, said to be emetic and purgative ; of *Bromus mollis*, reported to be unwholesome ; and of *Festuca quadridentata*, which is said to be poisonous in Quito, where it is called pigeonil. To these must be added *Molinia varia*, injurious to cattle, according to Endlicher ; and a variety of *Paspalum scrobiculatum*, called hureek in India, which is perhaps the ghohona grass, a reputed Indian poisonous species, said to render the milk of cows that graze upon it narcotic and drastic.

"It is, however, uncertain how far the injurious action of some of these may be owing to mechanical causes, which, in the case of the species of *Calamagrostis* and *Stipa*, seem to be the cause of mischief in consequence of their roughness and bristles. In their qualities the poisonous species seem to approach the properties of putrid wheat, which is known to be dangerous."

According to Riviere, the poisonous effects of the darnel are due to an acrid resin, which amounts to three-fifths of the ground seed. The plant grows abundantly in wet seasons, and in the green state is quite harmless, but the ripe seed is dangerous. According to Burkhard, the straw has proved deadly to calves. The poisonous properties of the lolium have been contested by some, and Weiss suggests that this might arise from the properties of the plant varying in different localities ; and the active principle is, without doubt, volatile ; so that old, dried, or roasted seeds are almost, if not quite, inert.

In man the seeds have produced heat, with pain in the stomach, nausea, vomiting, and diarrhoea, followed by languor, loss of vision, ringing in the ears, and vertigo, without proving fatal, though taken in a somewhat large dose. Mr. Tait of Melrose has recorded the following in the *Veterinarian* : "I was requested by a gentleman in this town to look at



three pigs that were taken suddenly ill. Before my arrival one of them had died. The other two were lying foaming at the mouth, with convulsive twitching over their bodies, and they also appeared to be dying. I lifted one up, when it immediately commenced running round about and against anything that happened to be in its way, or, meeting with an unyielding object, it stood thrusting its head against it; the other would not stand at all. We gave them some purgative medicine, but without any effect, for they soon died. I made inquiry of the man who fed them. He said that he had given them some dressings of barley a few hours before I saw them, in which was a great quantity of 'sturdy' (*Lolium temulentum*). On opening them, their stomachs and intestines were found to be highly inflamed. The lungs also were sadly congested. It is often remarked by old people that they were formerly in the habit of mixing the lolium with malt when brewing beer, as its intoxicating nature is very great; hence, I suppose, its name, 'sturdy.'"

Meyer observed horses, after having eaten of the seeds of this plant, to gather their feet under their body, foam at the mouth, have an anxious expression, irregular and quickened breathing, tucked-up appearance of the abdomen, pulse slow—from 25 to 28 beats in the minute—viscid saliva in the mouth, disturbed temperature of the body, listlessness, uncertain feeble gait. The condition lasted for about three days and three nights. A case is related by Seeger, which occurred in Switzerland. A horse had eaten such a quantity of the seeds of lolium, and was so stupefied and affected that he was taken for dead, and removed from the village. He awoke and returned to his stable, much to his owner's astonishment.

In olden times the darnel grass was the supposed cause of periodic ophthalmia, or moon-blindness. Seeger has experimented on dogs to ascertain the effects of lolium. Brosche has observed them in sheep, and has noticed the giddy and staggering symptoms; and after death no lesion of the alimentary canal, but inflammatory spots on the upper and right side of the brain, and on the right side of the medulla oblongata a tablespoonful of clear liquid. Rafn has found the lolium not injurious to fowls. Bauhin, Hertwig, Nestler, and others investigated the poisonous properties of darnel grass, and

Hertwig has fed sheep, horses, and hens with it for whole weeks, without seeing any ill-effects from it. This is confirmed by Professor Nestler's experiments on horses and cows. Nestler says "that we are quite certain of 100,000 cases in which the eating of *Lolium temulentum* has been unattended with mischief to one in which untoward symptoms were manifested."

#### FOXGLOVE.

The leaves and seeds of *Digitalis purpurea* possess active properties; the leaves only are officinal. The purple foxglove is a beautiful biennial or perennial hedge plant, indigenous in Europe, growing abundantly in the South of England. As a medicine, digitalis has been much extolled, from a marked and peculiar influence it exerts over the heart's action. It is capable of irritating the alimentary canal, is sedative to the nervous centres, and through them, affects the heart, as proved by the experiment of division of the parvagus, which prevents the digitalis inducing any effect on the central organ of circulation.

According to Delafond, the poisonous dose of digitalis for the horse is from  $1\frac{1}{2}$  to 2 ounces. Bouley and Reynal consider  $\frac{1}{2}$  ounce, and Hertwig 6 drachms, as sufficient to induce symptoms of poisoning. Half an ounce may excite gastro-enteritis. Cattle suffer from larger doses, and dogs die from the effects of from 2 to 3 ounces of the drug, if the œsophagus be tied. Six to eight hours after the exhibition of a poisonous dose of digitalis, there is dulness, loss of appetite, staring coat, injected visible mucous membranes, staring, prominent eyes, dilated nostrils, breathing and pulse accelerated; in the course of twelve hours, symptoms of gastro-intestinal irritation, with nausea, colicky pains, purging, and in some animals vomiting, supervene.

In about twenty-four hours the nervous centres are affected with symptoms of coma, fixed and haggard look, dilated pupils, feeble condition of the hind-quarters, skin and extremities cold, great muscular relaxation and debility, and death. The characteristic symptoms of poisoning by digitalis consist in violent and intermittent action of the heart; pulse feeble and indistinct; apparent mucous membranes of a violet

colour ; respiration accelerated, then becoming slow, irregular, interrupted ; rapid emaciation of body ; at first deficient urinary secretion, spasmodic efforts of the bladder, and, lastly, copious micturition.—(See Tabourin, “*Matière Médicale*,” p. 399.)

The *post mortem* appearances consist in marks of inflammation of the stomach and bowels ; black uncoagulable blood. The ventricles of the heart will probably be found remarkably contracted, and the auricles dilated.

Digitalis accumulates in the system, and for some time without obvious effects, but it may begin abruptly to act with great energy, as if with the accumulated power of all that may have been taken, and symptoms of poisoning become manifest.

In the treatment of cases of poisoning by digitalis, substances should be used containing tannin, as this renders the digitalia insoluble and inert. When there is much prostration, the system must be supported by stimulants until the poison is eliminated.

#### ARSENIC.

This is a metallic irritant poison, and is usually administered as arsenious acid. It may be swallowed or absorbed by the skin.

In small doses, administered daily for a considerable period, it induces chronic arsenical poisoning. This administration may be medicinal, and unfortunately too prolonged, or the arsenic may be accidentally taken with the food, on which it may have been deposited from factories or smelting-works ; or it may be absorbed by the skin in cases of mange, when black sulphur (*sulphur vivum*) is used as the parasiticide.

At first the small doses seem to improve the animal's health, but after a time the skin becomes affected, the hair or wool falls off, the skin may, if white, become blue, and there is a general loss of condition and all signs of unthriftiness.

Later on, if the floor of the abdomen beneath the stomach be pressed, there are signs of great pain, and in a few weeks an abscess forms in that neighbourhood, which ultimately bursts and leaves a fistula into the stomach ; the diameter of this fistula increases until ultimately food may escape by it.

Finally dysentery sets in and terminates the sufferings of the patient.

On *post mortem* examination the mucous membrane of the stomach is thickened, very red, and inflamed, and contains numerous hæmorrhages. The intestines are also much irritated and congested. In cattle and other animals, except the horse, there is frequently a gastric fistula. I have within the last few years seen cases of acute arsenical poisoning in horses.

In the first outbreak four horses ate of corn with which had been mixed a large quantity of white arsenic, and all these horses died; and in the other outbreak thirteen horses ate bran with which a large quantity of the poison had been mixed, and in this instance seven died, and the rest, though very ill for some time, ultimately recovered.

In each outbreak the symptoms were very similar. Violent colic from no known cause, great thirst, violent purging and straining, and fæces of a foul odour; pulse very rapid, respirations hurried, great anxiety of countenance, and a most marked yellow-red colour of the visible mucous membranes.

In addition to the above symptoms, in some cases there was great stiffness of the body, and partial loss of power of co-ordination, and in others again there was an acute laminitis.

*Treatment* consists firstly in relieving the acute pain with opiates, etc., and secondly in the administration of the freshly-prepared hydrated peroxide of iron, or of the carbonate of iron.

*Post mortem* examination reveals an acute gastro-enteritis, and chemical analysis shows, the presence of the arsenic, though in many cases of this acute poisoning the arsenic can be seen on or in the coat of the stomach.

A peculiar form of arsenical poisoning is known as the copper smoke disease, and is due to arsenic being given off from copper-smelting furnaces.

The chief symptoms are ophthalmia, general dulness, loss of appetite and condition. The gums are swollen and red, the teeth are encrusted with a bluish concretion, and these conditions are accompanied by marked ptyalism.

As the poisoning progresses the joints commence to swell,

and there is a general periostitis of the bones of the limbs. On *post mortem* the bones are found to be extremely friable and soft.

## PHOSPHORUS.

The form of poisoning by phosphorus which occurs in veterinary practice is of an acute character, and is usually due to the eating of rat poison by dogs and pigs, or of match-heads by fowls, the latter being quite a common method of getting rid of some noisy ones belonging to a neighbour !

The action of the poison is to cause a fatty degeneration of tissues by preventing oxidation, and to cause an acute gastro-enteritis, in some cases resulting in ulceration and perforation of the stomach. There is at first much depression, then exaltation of the nervous system and of the genital organs, great thirst, diarrhœa, and vomition of dark-coloured material which has a distinct odour of garlic, and is luminous in the dark.

Again, depression succeeds the exaltation, the mucous membranes become livid, and death shortly ensues.

*Treatment.*—Do not give oils, but give carbonate of magnesia, albuminous drinks, eggs, etc.

French turpentine is of great value, as it unites with the phosphorus and forms inert compounds.

On *post mortem* all signs of an acute gastro-enteritis are seen, phosphorus can be smelt, and the flesh and organs are phosphorescent in the dark.

## SALT (CHLORIDE OF SODIUM)—BRINE POISONING.

Most frequently seen in pigs which receive *souse* from hotels, and which contains the brine from corned meat, boiled ham, or pickled herrings.

This form of poisoning is very fatal, death usually occurring in a few hours.

In all animals the general symptoms are much the same—namely, great thirst, vomition, diarrhœa, epileptic fits, salivation, foaming at the mouth, and partial paralysis.

In addition to the above symptoms, in the pig we specially notice a diffuse red eruption on the skin, and a pinkiness of the mucous membrane of the mouth and eyes.

*Treatment* consists in the application of cold water to the head, acidulated drinks, demulcents and sedatives.

*Post mortem* reveals signs of an acute gastro-enteritis.

#### NITRATE OF POTASH—SALTPETRE POISONING.

Damp walls soiled with organic matter soon become covered with a white brittle crust composed of nitrate of calcium. The formation of the nitrate was once attributed to oxidation of nitrogenous materials. We know now that it is the result of the activity of a microbe, the nitrifying organism, the action of which in the soil is almost universal and necessary for the elaboration of organic manure, prior to their absorption by plants. These materials are absorbed in the form of nitrates, which are produced by the nitrifying germ at the expense of the ammonia, which results from the decomposition of nitrogenous matter. Now, the conditions for nitrification are abundantly realised in stables, the walls containing lime, the damp, the suitable temperature, the dirty walls soiled by urine and faecal matters containing much nitrogen. It thus happens that nitrate of calcium is continuously formed, and because it is not cleaned away it accumulates in sufficient quantity to induce poisoning in animals, which ingest it by licking the walls. Poisoning of this kind, noted by M. Buhl, veterinary surgeon, are common following the introduction of animals into stables that have long been occupied, and upon the walls of which much nitrate of calcium has accumulated. The symptoms observed are profuse diarrhoea, colic, weakness, fall of temperature, nausea, and vomition. There is a slowing of the circulation, and a purple hue of the visible mucous membranes.

As the effects become more marked the respiration becomes laboured, and in many cases bloody urine is passed, and the animal succumbs rapidly.

Treatment consists in the administration of stimulants, copious draughts of demulcents, and frictions and warmth to the skin.

#### SULPHATE OF ZINC

is sometimes, by accident or ignorance, mistaken for the sulphate of magnesia, and used for the making up of

“cleansing drinks,” or “purgative medicine” for cows, and acts as an irritant poison.

Shortly after administration the patient becomes nauseated, breaks out in a profuse perspiration, commences to vomit enormous quantities of fluid and food-stuff; the vomiting is unceasing, and drugs seem unable to check it, and ultimately—in fact, within a few hours—the patient dies of exhaustion.

The treatment is to give subcutaneous injections of morphia, and if possible to administer, tannin, oak bark, and demulcents.

## CHAPTER XLIX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES.

LOCAL diseases are those which have their primary seat and origin in any one tissue, function, or organ. They are sporadic diseases, and are generally, but not always, associated with constitutional disturbance, which, however, depends upon, and is secondary to, the original local affection.

#### (A.) DISEASES OF THE NERVOUS SYSTEM.

##### EPILEPSY—CHOREA.

Compared with the nervous diseases of man, those which affect the lower animals are few and insignificant. This can be accounted for in a variety of ways, but more particularly by the absence of the higher mental functions in the lower animals, and “in the way of life,” from their non-participation in the excitements, pleasures, hopes, and fears, as well as in the indulgences of the human race.

There are certainly some instances of what might be considered aberrations of intellect or morbid fancies in the lower animals, more particularly in the dog, by which the docile become ferocious, the good-tempered extremely irritable, the intelligent stupid or listless.

Animals also vary in degree of mental power: some are sharp, intelligent, easily taught, possessed of good memories, and are capable of manifesting a great degree of affection to those around them; while others are dull, hard to teach, have bad memories, and seem to live for themselves only. Notwith-



standing these varieties in the temperament and intelligence of animals, the purely mental—psychological—diseases are absent in our patients.

#### EPILEPSY.

A peculiar nervous state, difficult to define, but in which complete loss of consciousness prevails for a short time, associated with spasmodic contractions of the muscles, succeeded by debility, and sometimes by a desire to sleep. Epilepsy occurs amongst all animals, but is most commonly seen in young dogs.

The pathological condition of the brain which gives rise to epileptic fits is not yet determined; indeed, in fifteen out of twenty cases in which the brains of epileptic human patients have been examined, the structure of the brain has been found quite healthy. Occasionally, however, the brain and its membranes are actually diseased; thus the membranes may be found thickened, inflamed, or even ossified. In one horse, subject to epileptic fits, an abscess in the white matter of each hemisphere was found by me; in another, tumours in the choroid plexus; but in some instances it is due to some condition of the blood, as in those fits which are associated with catarrhal fever. In young dogs epileptic fits are associated with dentition, and with worms in the intestinal canal or stomach.

Dr. Brown-Séquard's researches into the origin of epilepsy are quoted at length by Mr. Gamgee in his *Domestic Animals in Health and Disease*, page 458. They are very interesting, but the tendency at present is to consider epilepsy certainly as a brain disease, but as one in which no definite pathological condition can in all cases be determined; and that the loss of consciousness, associated with excessive mobility, leads observers to regard those parts of the brain in the immediate vicinity of the *cella turcica* and basilar portion of the occipital region—for example, the central ganglia or medulla oblongata—as parts where, in future, morbid anatomy may yet discover a lesion.

*Symptoms.*—An animal in apparent health is seen to stagger and stare—the dog cries out at first, but is afterwards quite dumb—and then to fall into more or less violent convulsions, the whole system being agitated. The urine and fæces are sometimes passed involuntarily, the eyelids are closed, and if they are opened and the eyes examined, they will be seen to be in-

jected, sometimes convulsively agitated, at other times in a condition of strabismus (squinting), and sometimes fixed. During the fit the pulse is frequent, hard, sometimes intermittent, at other times scarcely perceptible; the respiration is stertorous, and sweats bedew the body.

I have carefully studied epilepsy, and have concluded that the condition termed vertigo or megrims is separate and distinct from what may be designated epilepsy, which is a disease, functional or otherwise, arising from extrinsic or intrinsic nervous irritation; whereas megrims is a term applied to all diseases which are associated with periods of insensibility or convulsions. I therefore limit the term epilepsy to a nervous affection characterised by convulsions, and which may appear either in the stable, kennel, or outside, at exercise or rest; whilst vertigo is applicable to a condition of syncope arising from disease of the heart or pericardium, and which occurs whilst the animal is at work or exercise. The epileptic subject, especially the horse, may be occasionally recognised whilst the animal is perfectly quiet, by a shaking of the head, and a working backwards and forwards of the ears, also by an occasional vacant, upward stare. This shaking of the head must not be confounded with that caused by ill-fitting bridles and head-stalls. Some horses are peculiarly square on the upper portion of the sides of the neck, and it will often be found that the borders of the neck are chafed by the head-gear. This should be carefully looked to before an opinion is given as to the soundness or unsoundness of a horse that shakes its head.

*Treatment of Epilepsy.*—If the fits are due to eccentric or peripheral causes—for example, to teething, worms in the stomach or intestinal canal, or indigestion, and this is not an infrequent cause, particularly in cattle—the first thing to be done is to remove these sources of irritation, by lancing the gums, extraction of offending teeth, and, by the use of vermifuges and purgatives, to expel worms or other intestinal irritants.

If the fits are due to disease of the nervous centres, all causes which may induce excitement or congestion of them are to be avoided; the food must be light and digestible, and the intestinal track cleared by means of purgatives, whilst the congestion, or a tendency to it, of the medulla oblongata and brain is best prevented or overcome by extract of belladonna. In the dog,

intrinsic epilepsy has sometimes given way to the salts of silver, zinc, or iron.

#### CHOREA.

*Definition.*—A series of automatic muscular movements, rarely seen except in the dog; clonic spasms, sometimes affecting the muscles of the face only, but generally involving those of the head, neck, and fore extremities; due to alterations in the spinal cord, or to disease of the facial nerve when affecting the face only.

The pathological conditions which give origin to the peculiar twitching or convulsive action of the muscles, characteristic of chorea or St. Vitus' dance, are not exactly determined, many pathologists who have investigated cases of the disease having failed to detect any definite morbid appearances. Consequently it has been regarded as an entirely functional disease, or as a disease due to an altered condition of the blood, the precise nature of such alteration not being known, and also as a symptom of some diseases of the heart.

In the dog, it may with good reason be said to be first due to an altered condition of the blood, generally brought about by, or giving origin to, the disease called distemper, of which it is so often a distinguishing feature; whilst in the horse, spasmodic contractions of the thoracic and cervical muscles, of the nature of chorea, are often associated with cardiac diseases of a rheumatic type.

*Symptoms in the Dog.*—The convulsions may be confined to one fore leg or shoulder, of which there is a peculiar jerking action, like a series of pulsations. At other times both fore legs are affected, when there is a nodding or depressing of the head and neck. In many cases it is restricted to a constant jerking of the head and lower jaw, with marked twitchings of the superior cervical muscles; the muscles of the eyelids, and sometimes the muscles of the eye itself, are also affected. The convulsions do not cease whilst the animal is lying down, but they generally, although not uniformly, cease during sleep. In some cases the dog will remain otherwise healthy for an indefinite period; but if the chorea attacks it whilst in a debilitated state, the expenditure of muscular action, and the general restlessness, slowly undermine the remaining strength; the dog becomes emaciated, subject to fits, and finally paralysis and death supervene.

The *post mortem* appearances are very indefinite. If succeeding a recent attack of "distemper," redness and softening of the spinal cord, meningeal dropsy, and in some cases spots of congestion on the medulla, optic thalami, and corpora striata, are discoverable; whilst in cases which have become chronic no lesions have been discoverable after death. Most probably, however, some change has taken place in the composition of the nervous system, which has not been detected; and this supposition is supported by the evidence that in acute or recent cases morbid changes are usually detectable. In some cases the disease disappears spontaneously, more especially if mild, and the patient be moderately strong.

In the horse chorea may usually be regarded as a concomitant of other diseases, and will be mentioned in its proper place.

*Treatment in the Dog.*—Attention to the condition of the bowels, removal of all sources of irritation by mild purgatives, generous diet, and tonics.

In human medicine, Dr. Walshe recommends the extract of *cannabis indica* as the most efficacious remedy, in doses of one-fourth of a grain thrice daily. In other cases much benefit has accrued from the cold bath. In my own practice I have found that camphor and spirits of nitrous ether are sometimes very useful, allaying the restlessness and diminishing the convulsions. At the same time the animal strength has been improved by good food and sulphate of iron. I find that the best diet for dogs is milk and oatmeal porridge in the morning, and an allowance of flesh, according to size, in the evening. Bromide of potassium or chloral hydrate, oxide of zinc, or arsenious acid, have been recommended.

The foregoing diseases not being essentially due to inflammation, congestion, or organic alteration of any particular part of the cerebro-spinal system, I have placed them under the general head of "Diseases of the Nervous System." Having done this, I now purpose giving a detailed description of those nervous diseases to which a locality can, with some certainty, be ascribed.

## CHAPTER L.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (B.) CEPHALIC DISEASES.

##### INFLAMMATION OF THE BRAIN SUBSTANCE—CEREBRITIS.

THIS is a very rare affection, and its occurrence can only be proved by *post mortem* examinations. Mr. Gamgee says that it is impossible in practice to distinguish between inflammation of the brain—cerebritis, and inflammation of its coverings—meningitis. In this he is so far correct; but pure cerebritis does occur, if we are to look upon the formation of abscesses in the white substance of the cerebrum as evidences of inflammation.

I have seen two cases which presented this appearance *post mortem*, and another, that had all the symptoms of cerebral abscess, and recovered, but was peculiar in its motions and action, had epileptic fits, was unable to walk, turned with difficulty, and moved its limbs automatically as long as it lived.

The three cases mentioned were suffering, or were recovering from strangles, the advent of the brain symptoms succeeding to the formation of the abscesses in the intermaxillary space. In the two that died, the symptoms were, great drowsiness, slowness of the pulse, inability to masticate food, although a desire to eat still remained, staggering gait, dragging of the feet, and subsequently coma, with stertorous breathing, amaurosis, paralysis, embracing the muscles of the head, trunk, and extremities, rapid sinking, and death. In one case the voluntary muscles were quite flaccid, and were incapable of any movement; whilst in the other some contractile power still remained, and now and then feeble struggling would take place. It was able to move

the limbs, but very slightly, and possessed a little sensibility when pricked with a pin. In both cases the sphincters were relaxed, and urine flowed involuntarily.

In the third case—the one which lived—the symptoms were those of incomplete paralysis of the whole muscular system; there was drooping of the lips, semi-closure of the eyelids, staggering gait, automatic muscular movements, and when the horse was moved, muscular action continued for some time. When its head was pressed against a solid object, such as the walls of the stable, it would continue to move its limbs, and press its head and body forward with great force; indeed there seemed to be a tendency to go straight forward. The desire for food was pretty good, and although mastication and deglutition were performed with difficulty, it was able to take a fair amount of support, consisting of milk, eggs, gruel, &c., in addition to ordinary food. It continued in this condition for about fourteen days, when it burst through the box door during the night of 31st December 1862, and was found prostrate, and covered with snow, in my yard in Bradford, when the groom went into the yard on the morning of 1st January; it was immediately dragged into its box, and covered over with clothing and straw. It lay prostrate, steaming with perspiration, thus induced, for three days, during which time it scarcely moved. As the bowels were constipated, a full cathartic was given, which responded on the second day; the urine flowed involuntarily, doing away with the necessity of using the catheter. On the third morning some degree of consciousness returned, and it was now able to drink water—small quantities of which had been carefully given it out of a bottle—and in the evening it rose to its feet. It now gradually recovered, but ever afterwards showed some degree of aberration of mental powers—"intellect," if the term is proper—and was always called the "cranky horse" by the men about it. I conclude that this was a case of small abscess in the cerebral substance, from the fact that it was at the time affected with a pyogenic disease—strangles—and from the similarity of the symptoms to those seen in the cases in which I had the opportunity of making *post mortem* examinations; and I account for the partial recovery on the supposition that the pus had either broken down and was absorbed, or had become inspissated, and the medullary tissue of the brain accommodated to its presence.

The differences between cerebral and meningeal diseases are arranged as follows by Aitken, and as, to some extent, they may prove useful in diagnosis, I take the liberty of quoting them:—

## CEREBRAL DISEASE.

1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, such as paralysis, anæsthesia, loss of memory.

2. Cerebral disease is not commonly attended by high-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia, or tenderness.

3. Little vascular excitement attends cerebral disease, nor is there frequently any highly marked general disturbance.

4. Paralysis and anæsthesia, losses of volition, ideation, perception, and the like, characterise cerebral disease.

## MENINGEAL DISEASE.

1. It is not till some time after the detection of signs of disease that diminution or loss of nervous function takes place.

2. The subsequent diminution or loss of nervous function which succeeds the prolonged existence of "head symptoms" is generally preceded in cases of meningeal disease by extremely severe excitement or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.

3. In meningeal affections there is usually much local vascular excitement, with general disturbance.

4. Spasms, or convulsions, pain, and delirium are the general features of meningeal disease.

In all the three cases of cerebral abscess mentioned, there was the absence of the above-mentioned indications of meningeal disease; and indeed the *post mortem* of the two fatal ones proved this, and even the automatic movements of the limbs of the third case clearly proved that volition was to a great extent destroyed.

In each of the brains examined, the abscesses were symmetrical, situated in the anterior lobes of each hemisphere, and were seen, after the removal of the dura mater, as two bulging prominences, measuring about an inch in diameter, which, when opened, gave exit to a thick, whitish pus, of good consistence and free from smell. The walls of the abscesses consisted of the

medullary substance, covered here and there in one case with small red points; whilst in the other the surrounding substance was of the natural colour.

#### TREATMENT.

But little can be suggested in the way of remedial treatment. If the animal lives sufficiently long, the *liquor puris* may be absorbed, and the shrivelled pus cells remain, as in the case recorded. But little assistance can be given to this, beyond keeping the bowels open by an occasional cathartic, to act as a derivative, and attending to the diet of the animal; the food in all instances must be nutritious but non-stimulating. Care must also be taken that the animal does not injure itself by falling; and to prevent injury to the head, some soft substance, such as a sack stuffed with straw, should be placed in front of the patient, in order that it may rest its forehead upon it.

#### MENIÈRE'S DISEASE.

A series of symptoms indicative of irritation in the neighbourhood of the ears and poll—which irritation may be intrinsic or extrinsic—is named after the author who first endeavoured to elucidate the disease.

I have been fortunate enough to have seen three cases, which I looked upon as similar in symptoms to *Menière's disease*.

The attack usually comes on suddenly, and when the animal is being harnessed or out at work. In some cases the patient shakes its head most violently from side to side, and upwards and downwards, perhaps unceasingly for a minute or more, and then becomes quiet, as if it had only been a temporary irritation such as might be caused by the bite of the inside of the ear by a fly or by an acarus, or a maggot—as often seen in sheep; but on examination, none of these can be discovered; and perhaps in a few hours, days, or even weeks, there is another unaccountable attack.

In a patient I have at present under observation there is a dislike to sudden bright light. The animal shows no sign of disease on dull days, or in a darkish stable; but



a sudden bright light, or working in the sun, causes it to show peculiar nervous affection of the head, a tendency to stagger, but not falling completely down. In this case the animal is at once relieved by a fit of coughing and sneezing, as if the irritant were in a nostril, or perhaps in the pharynx.

In another case I had the horse was worst when being bridled or unbridled, and seemed to try and shake its bridle off; it also showed this irritation after standing outside for some time, but it only became slightly giddy.

Some say the disease is due to abnormality of fluid in the semicircular canals of the ears—perhaps a deficiency in increase in quantity or an alteration in quality. Others say it is indicative of brain disease. Whatever be the cause, it is well to remember that it *might* be a movable one, such as acariasis of the ear; an irritable condition of the mane at the poll; perhaps too long a forelock, and some of its hairs irritating the eyes or tickling the ear; or it may be an ill-fitting bridle, or too heavy a one. It may be simply stomachic, and so easily treated.

I am inclined to think that in some cases the cause of the symptoms may be found in the guttural pouches.

## CHAPTER LI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

- (C.) ENCEPHALITIS, INFLAMMATION OF THE BRAIN AND MEMBRANES—CEREBRO-MENINGITIS, PHRENITIS, MAD STAGGERS, PHRENZY, COMA, SLEEPY STAGGERS, STOMACH STAGGERS, ABDOMINAL VERTIGO, &c.

*Definition.*—A morbid condition of the brain and its membranes, attended with congestion of the vessels, effusion into the ventricles and subarachnoid space, with exudation of lymph between the convolutions, and arachnoid and pia mater.

Percivall, Blaine, Dick, and others describe two forms of staggers—the one arising from inflammation of the encephalon, and the other from engorgement of the stomach, respectively denominated “mad” and “stomach staggers;” and we are led to conclude that in the latter disease the brain is only sympathetically affected, or is disturbed by reflex irritation. Mr. Gamgee says, “I regard phrenitis as almost an unknown disease.” If by this Mr. Gamgee means the phrenzy or mad staggers of our forefathers, experience compels me to agree with him; but if he intends to convey the idea that encephalitis, or inflammation of the brain and its membranes, occurs as a traumatic disease only, I am bound, by the same experience, to differ from him, as experience amongst the living, and examinations of the dead subject, have convinced me that the conditions named “mad,” when not caused by violence, traumatically, or by tumours, &c., and “stomach staggers,” are symptomatic of but one and the same malady, namely, inflammation of the brain and its meninges, and that the varying degrees of violence, convulsions, frenzy,

stupor, coma, immobility, or paralysis, depend upon the seat of the congestion or inflammation at the time such symptoms are manifest. For example, the coma and loss of function become apparent when the brain proper is congested, or when pressed upon by effusion; the convulsions, tetanic spasms, and frenzy, when the pressure is not so great, and when, in all probability, the membranes are the seat of the congestion or irritation; and paralysis of a part of the body, generally the posterior extremities, when there is inflammation of the spinal cord and its membranes.

There are many pathological conditions of the brain which give rise to symptoms of frenzy, such as tuberculosis of the membranes, tumours, some non-cognisable morbid poisons, uræmic poisoning, rabies, the irritation of melanotic deposits, &c.; but inflammation gives rise to loss of function, and instead of the exalted condition, so graphically described by Percivall, we have in true cerebritis stupor, coma, and general paralysis.

Mr. Gamgee describes a form of encephalitis in horned cattle arising from alcoholism. The cases are reported by Mr. George Dundas in the *Edinburgh Veterinary Review*. "The disease is due to a practice, prevalent in some parts of Scotland, of giving "burnt ale" to cows, in the neighbourhood of distilleries. The ale is given by steeping straw in it; and the animals will also drink it freely. They often sleep soundly after such a beverage, and sometimes symptoms of intoxication are manifest. The symptoms are as follows:—The head is turned singularly to the side, and is slightly elevated. The pupils are widely dilated, and the eyes have a remarkably wild appearance. On approaching the animals, they wink rapidly, and tremble. There is marked heat of head, horns, and ears. When pressed with the finger in the axilla they fall instantly, and when pulled by the head they incline to turn over. The pulse is about seventy or eighty per minute."

"After death, all the organs are found healthy except the nervous centres, and both brain and its membranes are found congested. This congestion often extends into the spinal canal, and the pia mater over both the brain and cord is the seat of red spots. The redness is either ramified, or is obviously due to blood extravasation. Clots of blood have been found in the lateral ventricles, and around the spinal marrow in the cervical

regions. There is evidently softening of the brain substance as a direct result of this condition.”—(GAMGEE’S *Domestic Animals in Health and Disease*.) I have never witnessed this effect of alcohol on cattle, although I am familiar with the fact that the “dreg” from distilleries is largely used for feeding purposes, and that it is an excellent adjunct to other foods.

The most common form of congestion of the brain and its membranes, both in the horse and horned cattle, is that called stomach staggers, sleepy staggers, or grass staggers—a disease which sometimes rages as an enzootic.

#### SYMPTOMS.

The animal at first appears dull, listless, falls asleep whilst standing, or drowsily nods its head; eats slowly and at intervals; the breathing is generally slower than is natural, sometimes slightly accelerated, but it is always of a somewhat snoring description. The pulse is also slower than natural, from twenty-six to thirty, full and rolling. The animal walks with a straggling gait, staggers, and seems as if about to fall. If suddenly disturbed whilst in the somnolent condition, it looks around excitedly, shivers violently, and seems affrighted; but soon becomes calm again, and may remain so for a short period, especially if kept in a dark, quiet place. It now and then thrusts its head against the rack or wall of the stable; moves the limbs automatically; rears, hangs back, and breaks the halter, or gets its fore feet into the manger, and elevates the nose high up into the rack. The eyes, mouth, and rectum are injected, and of a yellow tinge. In some cases, the yellowness of the mucous membranes is a very prominent symptom. As the disease advances, the extremities become alternately hot and cold; sweats bedew the body; there will be twitchings or clonic spasms of the superficial muscles of the neck, breast, and hind quarters. Violent convulsions will now occur; the whole body becoming stiff and rigid, and the respiratory movements extremely difficult. During these spasmodic—tonic—attacks, the tail will be elevated; the membrana nictitans drawn over the eye, as if the animal were suffering from tetanus, and the pulse frequent, hard, and wiry, the eyes fixed and amaurotic, the mouth clammy, and the urine may be ejected by a convulsive

effort. All at once the tonic contractions subside, and there will be great muscular debility; the legs bend, the animal totters, sometimes falls, and when down will fight convulsively, and for a time be unable to rise. By slow degrees consciousness to some extent returns; the animal may then regain its feet, and will perhaps commence to feed; then fall asleep, with its mouth full of food, or look about in a wild, staring, vacant manner, or ramble unconsciously about the box, striking its head against everything that may come in the way, the eyes amaurotic, and the animal quite blind.

Sometimes there is flaccidity or paralysis of the muscles supplied by the cranial nerves, the lips are pendulous, and the tongue hangs out of the mouth. At other times there will be tonic spasm of the facial and masticatory muscles, and some degree of trismus present.

The disease may attack the animal whilst at grass in the field. It is then observed to ramble about in an unconscious, drowsy manner, until it meets with some solid object, against which it fixes its head, and then moves its limbs continuously.

In cases which have been noticed at the commencement of the attack, some degree of rigor has always been observed premonitory to the development of other symptoms.

The above symptoms are subject to some variations. In some animals the comatose and paralytic conditions are the most prominent throughout, whilst in others delirium, convulsions, and spasmodic contractions alternate with the stupor. In other cases, again, the spinal system seems most affected when paralysis, without loss of consciousness, or with but a slight degree of drowsiness, is present.

There appears to be a general diminution of the various secretions; the costiveness is obstinate; the urine is secreted in small quantities, and is particularly high in colour. The function of the liver is also suspended, and the whole system becomes tinged with the non-excreted biliary colouring matter—biliverdine.

As the disease advances the coma is more profound, or the fits of excitement and frenzy more frequent, and of shorter duration, leaving the animal more and more enfeebled; the pulse is now small and quick, and the breathing stertorous and difficult; profuse sweats bathe the body, the sphincters relax, the

animal is unable to stand, and dies fighting convulsively, or in a state of profound coma.

#### ETIOLOGY.

It is very generally held by writers upon veterinary medicine that the malady arises from an over-loaded and impacted condition of the stomach, that the cerebral symptoms are purely reflex or sympathetic, and that no actual disease of the brain or its meninges is present. These assertions are not supported by the results of investigation into the morbid anatomy of the malady, further than that the stomach has been generally found filled with food. Now, if engorgement of the stomach were the cause of the train of symptoms seen in this malady, then coma, delirium, or paralysis would be general in the majority of cases of engorgement, seen in the routine of general practice, but this is not the case. Numerous instances of engorgement, impaction, even to rupture of the stomach, constantly come under the notice of the practitioner, but signs of any brain affection scarcely ever occur. We must then look to something more than mere impaction as the cause, and I think this will be found in the *nature* of the food.

In Scotland it has been called "grass staggers," from the fact that it occurs when the animal is fed on green food; but ordinary green food does not induce it. I have very carefully noted every case which has fallen under my care for some years, and I find that grass, more particularly rye-grass, when it has commenced to ripen, or when it has been cut, and allowed to heat and ferment before being used, is a fruitful source of this disease. I find that my observations are borne out by those of Professor Dick and others, and that not only horses and cattle are liable to the disease from this cause, but sheep, and especially lambs.

Mr. Brydon, V.S., Traquair, in a letter to Professor Dick, says that lambs are often destroyed by eating the tops of rye-grass; and that he has found the tops of rye-grass two or three inches in length in their stomachs, causing inflammation.

Professor Dick again says—"From what has been stated it will appear that, when rye-grass begins to ripen, a change should be made in the food by placing the animals on other pasture. The grass should be cut before it has quite ripened, as it will be found in that state innocuous." He then refers to a statement made

by White of Exeter, in White's *Farriery*, that the disease occurred in one farm in South Wales, from hay made the previous year (1800), and concludes that "it seems more than probable the hay had been over-ripe when made, and that the process of withering had not destroyed the irritating or noxious, or perhaps narcotic, quality of the over-ripe grasses."—(*Veterinary Papers* by Professor DICK.)

I quite agree with Professor Dick that the disease is due to some peculiar narcotic principle, that is developed in the grass at this time, or which may be developed by the process of heating and fermentation, when cut at an earlier stage of its growth; for every-day experience proves to us that food, even rye-grass included, has no effect in producing symptoms of cerebral disturbance, when used in its ordinary condition, and that it is only when in a transition stage, as it were, between grass and hay, that it seems to possess toxic qualities.

After careful observation I have arrived at the conclusion that the seat of this disease is in the brain, the spinal cord, and their meninges; that, owing to the quality of the food, a degree of narcotism is first produced, speedily succeeded by congestion and other changes to be described in the morbid anatomy, and that owing to this derangement of the great nervous centres, paralysis of the digestive apparatus is the result, and the stomach becomes sometimes engorged, from the fact that the animal continues to feed, when the digestive as well as other functions are in abeyance.

This disease seems to have first attracted notice in 1787, the summer of which was hot and dry. It raged in the south-west of England and Wales in 1800 and 1819, the summers of which were also hot and dry, prevailing most commonly amongst horses at grass in low, wet pastures, where the grass was rank. It was supposed to arise from their eating ragwort or staggerwort—*Senecio Jacobæa*—a plant supposed to contain a poisonous principle, or some other poisonous herb; but of this there is no absolute proof. We have, however, sufficient evidence to prove that it originates when animals eat rye-grass in the condition described. Mr. Gamgee says the disease may arise from gastric derangement, brought about by eating wheat, or even oats and bran, in large quantities. My experience convinces me that this is not the case, and that mere over-loading is more apt to pro-

duce rupture, tympanites, inflammation of the bowels or feet, or intestinal apoplexy.

All kinds of damp fodder soon become covered with rusts or moulds. Amongst the former, the *Puccinia graminis uredo*, or red rust, has induced disease in rabbits, characterised by gastric irritation, vertigo, and convulsions (Frank). There are also black moulds—microphytes—closely related to yeast plants, indeed said to be a less advanced stage of the yeasts, and these are now proved to cause the formation of ptomaines, having a deleterious effect upon cattle, manifested by symptoms of depression, abdominal pains, constipation, succeeded by a foetid and sometimes bloody diarrhoea, bellowing and groaning, some degree of tympanites, and sometimes paralysis of the tongue and pharyngeal muscles, with continued movements of the jaws, salivation, and cough. These latter symptoms are more particularly due to black rust—*Tilletia caries*. These moulds and rusts are not uncommon on ill-dried feeding cakes, and are undoubtedly a source of much mischief.

#### MORBID ANATOMY.

From various dissections, very carefully made, it is found that impaction of the stomach is but an occasional complication. In many cases some degree of congestion of the villous coat has been present, and this appearance led Blaine to conclude that the disease was a specific form of gastritis; but this is not an invariable lesion, and is most probably as much induced by the action of medicinal agents as by food. The stomach may be quite empty; sometimes it contains some amount of food, and at other times it is impacted; there is always a congested condition of the lungs, as is the case in death by coma. The brain and its membranes are invariably congested; the former, after removal, seems to be in a swollen condition; the dura mater seems stretched, and the convolutions appear broader and flatter than natural, as if they had been pressed against the cranial walls. The vessels of the pia mater are injected and tortuous, and that membrane itself is easily lacerable, and may be stripped from the surface of the convolutions without tearing the cortical substance, which of itself looks darker than natural. On cutting into the brain both grey and white matters are



studded here and there with red points. The plexus choroides is large, highly injected, sometimes covered with a thin film of exudation, and the lateral ventricles are filled with fluid.

In the spinal form of the disease, namely, that characterised by paralysis of the hind extremities, the congestion and effusion are generally limited to the spinal cord and its membranes in the dorso-lumbar region; the arachnoid space is filled with serosity of a reddish colour; the pia mater is congested, and the cord itself in a red, softened condition.

*Prognosis.*—In the horse the probabilities of recovery are much greater than in oxen and sheep, in which it is very fatal. The recovery in the horse, however, has been slow and protracted, some degree of paralysis in many cases remaining for several weeks after the acute symptoms have passed off. Recovery may be expected when the symptoms do not become much aggravated during the first day or two, when the delirium is not excessive, and when the coma is not continuous and profound. In the spinal form the animal may fall and be unable to rise, very early in the disease; this is but of little moment, provided the animal does not struggle much, and work itself into a state of fever and exhaustion. In the cerebral form, inability to retain the standing posture is always a bad sign, more especially if the delirium be continuous. The age of the animal must also be taken into consideration; the young and strong may recover, the old and debilitated succumb.

#### TREATMENT.

In the early stages, when the pulse is full, and has tone, bleeding has a beneficial effect, by directly unloading the vessels of the brain. Indeed, I have witnessed most marked results from this, the almost unconscious, amaurotic animal regaining its sight and consciousness in a short time. Four, five, six, or even eight quarts can be taken from the jugular with advantage. If, on the contrary, the pulse be feeble, accelerated, as in the later stages of the disease, bleeding only hastens the animal's death. A smart cathartic is always to be administered, and no danger need be apprehended from superpurgation. The purge may be composed of from six to eight drachms of aloes, and a

drachm of calomel for the horse; twenty ounces of sulphate of magnesia, and fifteen croton beans for a full-sized ox or cow; after which the animal is to be kept as quiet as possible; all food must be removed; plenty of water allowed to drink; an enema now and then administered; and the head bathed with cold water. The attendant is to do all this quietly and calmly; there must be no shouting, or any noise to disturb the animal, nor is it to be continually dosed with medicine. Time must be allowed for the purgative to act, and in some cases there is no response for two or three days; but if the symptoms abate, no alarm need be felt, as the bowels are always slow to act when in a paralyzed condition. Should the symptoms increase in severity, belladonna might be tried, in two-drachm doses of the extract, or half-grain of atropine injected subcutaneously.

I think this form of constipation offers a fair field for the chloride of barium, about ten grains of which might be intravenously administered to induce rapid action of the bowels.

When the acute symptoms have passed off, it may be necessary to apply blisters to the loins, or at the back of the poll; but during the earlier stages, more especially if the horse is restless, nothing should be done to irritate and annoy it, or it may work itself into a state of excitement which speedily terminates in death. In addition to blisters, I have found the iodide of potassium, given twice a day, to be exceedingly useful in promoting the absorption of any exudate or effusion; and should the bowels remain torpid, and an imperfect paralysis continue, *nux vomica* may be added with advantage. It may be here remarked that the arrestation of the urinary, biliary, and intestinal secretions are results of cerebral disturbance, and that when this is removed the secretions regain their natural condition. Purgatives are useful as removers of the cause, which is food containing deleterious principles, and as derivatives. If the pulse be feeble, small doses of stimulants—ammonia—may be administered. I am, however, no advocate for large doses of stimulants, as they tend to excite the animal and make it restless, when quietude is of the greatest importance.

## CHAPTER LII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (D.) CEREBRAL APOPLEXY.

*Definition.*—"A disease essentially characterised by the sudden loss, more or less complete, of volition, perception, sensation, and motion, depending on sudden pressure upon the brain (the tissue of which may be morbid), originating within the cranium."—(AITKEN.)

Apoplexy is of two kinds:—1st. That arising from degeneration of the cerebral vessels, with rupture of them, and extravasation of blood upon or within the substance of the brain; and 2d. That in which congestion of the cerebral vessels—not of themselves necessarily diseased—is the primary condition; this, when excessive, resulting in rupture and extravasation.

#### SEMIOLOGY.

*Apoplexy from the rupture of degenerated blood-vessels.*—There may be some premonitory symptoms, such as staggering and partial paralysis, but generally the animal falls suddenly without warning. After falling it may lie prostrate, in a state of unconsciousness, without the power of voluntary motion, perfectly insensible to surrounding objects, and dead to all ordinary feeling, with its eyes wide open and presenting a ghastly stare, the pupils dilated and insensitive to the light—amaurotic. The breathing is stertorous, the pulse small, rapid, and thready, the surface of the body cold, or bedewed with a cold sweat, the limbs flaccid, the mouth open, and filled with frothy saliva, and in some severe cases the sphincters are relaxed. In other

instances the animal may still retain the power of muscular movements; but they are irregular, and intermixed with spasmodic contractions. Whilst down, it fights convulsively, presses the back of its head violently against the wall or other solid body. Some degree of opisthotonos is present, the back is arched downwards, and the hind legs extended backwards. The eyes move about convulsively, or there may be persistent strabismus of one or both of them; the pupils may be dilated or contracted, alternately contracted and dilated, or they may be natural, or one may be contracted, and the other natural or even dilated. The respiratory movements are sometimes spasmodic; now and then there may be a stertorous sound; at other times sighing, and sometimes expressions of great pain, as if the animal had been sharply wounded with a cutting instrument.

These symptoms may alternate with intervals of quietude, when the animal will fall into the comatose condition. The pulse may then fall below its natural standard, and the respiratory movements may become slower than natural, with heavy, deep inspirations. The various secretions are, in all cases, suspended, and the animal gradually sinks from increasing brain pressure, or suddenly from renewed extravasation.

The symptoms of apoplexy present some degree of variety in their character, and in the course of experience one cannot but remark that conditions which were laid down by our teachers as being invariably present, are sometimes altogether absent, or are present in a very modified degree. For example, it is generally taught that when a patient is apoplectic and comatose, the pupils are widely dilated, and the eyes amaurotic; but this is not always the case, neither is contraction of the pupils an invariable sign of brain disease. In many cases the condition of the eyes is quite normal. Again, the loss of consciousness varies very much; in one case we have complete insensibility, in another a degree of hyperæsthesia to touch and sound, as expressed by convulsions and tremors, when the animal is touched or spoken to. In one case, although a large clot, extending from the superior surfaces of the crura to the base of the brain was discovered *post mortem*, it was observed that when left alone the horse would lie more or less quietly; immediately it was disturbed it would fight convulsively, the breathing would become spasmodic, and it would dash its head with

violence upon the ground; when pricked with a pin it showed extreme agony.

*Stertorous breathing.*—This symptom, like the other, is not invariably present, indeed it is often absent in undoubted apoplexy. At all times it indicates severe lesion, and that the pons varolii or medulla oblongata are implicated.

#### MORBID ANATOMY.

Apoplexy from degeneration is seen in old horses, in which the *post mortem* examination reveals the cerebral arteries in an atheromatous condition. The first attack generally proves fatal, but I have seen two cases of relapsing apoplexy, in which distinct *post mortem* traces of former extravasation were found.

The clots are either superficial or deep seated. Superficial clots are generally supposed to be due to external violence. This is, however, not always the case. Extravasations are found in the lateral ventricles, from a rent in the corpus striatum, or from rupture of some of the vessels of the choroid plexus, upon the surface of the cerebrum, and at the base of the brain; in one instance there was, in addition to a tumour, extravasation from degeneration and rupture of the vessels of the falx cerebelli.

The extravasations are occasionally circumscribed, but most commonly diffused, and spread over a more or less extensive surface, extending from the lateral to the fourth ventricle, and sometimes surrounding the crura cerebri.

In rare instances the symptoms may take some days before they become fully developed. This is due to very slight but increasing hæmorrhage. In the majority of cases the attack is sudden, and the loss of power almost instantaneous. Congestion of the brain—as in the last described disease—occasionally terminates in apoplexy, or rupture of the cerebral vessels and extravasation of blood; but congestive cerebral apoplexy is a rare disease in the horse, that which is usually met with being due to degeneration of the vessels.

## CHAPTER LIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (E.) APOPLEXY FROM CONGESTION.

THIS form of apoplexy is of frequent occurrence in cows, but occasionally occurs in goats and mares, and is variously termed parturient apoplexy, milk fever, or dropping after calving.

*Definition.*—A parturient disease, characterised by suppression of the lacteal secretion, congestion of the brain, and apoplexy.

#### ETIOLOGY.

Various opinions are held upon the causes and origin of parturient apoplexy, some writers maintaining that it is a blood disease, whilst others state that conformation or anatomical peculiarity is sufficient to account for its occurrence in the cow, and its absence in the mare. The late Mr. Barlow, in a most profound and remarkable paper, traced its origin to derangement of the great sympathetic nerve, with consequent arrest of secretion, resulting in general congestion, which became localized in the brain and spinal cord; whilst other observers express an opinion that the disease results from derangement of the digestive apparatus, and that it is a peculiar form of indigestion.

The anatomical theory is upset by the fact that the disease is inseparable from domestication and stimulating food; peculiar to deep milkers, and scarcely ever succeeding difficult parturition, which most assuredly would be the case if shortness of neck, excessive—natural—vascularity of the brain and its membranes were predisposing causes.

The other theories, namely, that which traces the origin of the disease to derangement of the function of the ganglionic or sympathetic nerve, and that ascribing it to indigestion, may conjointly, I think, be looked upon as offering part of an explanation. Within the past few years it has, however, been clearly proved that the disease in cows is due to the formation of a certain ferment or ptomaine, or toxin (as it is variously described), in the udder—that this poison is absorbed, and has a specific action on the nerve centres.

Parturient apoplexy rarely occurs prior to the third period of parturition, but I have seen it, in two instances, succeed the second calving. Cows of all breeds are subject to it, provided they are deep milkers. It seldom attacks cows in which the lactiferous system is not highly developed, no matter of what breed they be. Age, and the profitable property of giving an abundant quantity of milk, are acknowledged and well-known predisposing causes; so much so, that dairymen very often decline to buy a deep milker immediately prior to a third or subsequent parturition. This tendency to the malady is promoted and intensified by warm weather and stimulating food, which is generally allowed in great abundance. All cows, but more particularly those which are profitable milkers, ought to be well fed; but it should be remembered that prior to the act of parturition, deep-milking cows, which are dry or nearly so, rapidly become plethoric, and that this state of body, at the time when the calf is born, is one which very commonly excites this fatal malady; hence, in feeding an animal of this kind, great care should be taken that the food be not over-abundant, too highly nitrogenous, or too watery. A previous attack of parturient apoplexy also predisposes to a second, and generally fatal attack of the malady.

The exciting causes may be looked for in the act of parturition itself, mistake in the dietary, and in the season of the year.

In rare instances, parturient apoplexy occurs during or immediately prior to the birth of the calf; but almost invariably the first symptoms are not manifested before the lapse of some hours—from twenty to twenty-four hours is a very critical period—or even two or three days after parturition.

When symptoms of the malady are not manifested for some time after the birth of the calf, it will generally be found that

the secretion of the mammary gland has been in an average, or even abundant quantity, that the appetite has been good, and rumination naturally performed. Premonitory to other symptoms, it is generally seen that the secretion of milk is suspended.

The arrest of the lactiferous secretion is doubtless due to the disturbance of the organic system of nerves.

In the natural or healthy condition, the blood required for the support of the foetus *in utero* is diverted after the parturition into the mammary gland, for the purpose of supplying material for the formation of milk. One portion of the ganglionic system is thus brought into increased action, whilst that which during the pregnancy had been developed in the uterus, being no longer required, falls into a condition of at least temporary atrophy.

#### SEMIOLOGY.

In many instances the symptoms appear suddenly, and with great violence, the disease running its course, and terminating fatally in a short period, but most commonly there are some premonitory signs. The secretion of milk is stopped, the cow hangs its head, ceases to feed, and paddles with its hind feet. By-and-by the breathing becomes hard and rapid; it sways from side to side; the hind legs double at the fetlocks, and at last it falls. The eyes are blood-shot, in some cases wild and staring; the eye-lids twitch spasmodically, and an abundant flow of tears runs over the cheeks. The ears, horns, and forehead are now intensely hot; the animal lies either in a state of perfect stupor or coma, or dashes itself violently about; the head is thrown from side to side, and there is danger of the horns being knocked off.

In some cases the nose rests firmly upon the ground, and if the head is lifted up, it will fall back like some lifeless body; at other times the head is brought back to the side, where it remains firmly pressed against the shoulder or neck. There is often a disposition to lie upon the side, in which case the neck is stretched out, and the limbs extended, the eyes glassy, and the mouth open; tympanites soon sets in, and the cow will die in a very short time if its position be not altered. Very early in



the disease the power of vision is lost, the eyes being amaurotic ; the power of swallowing is also lost, or very imperfectly retained. The cow may remain in this condition for several hours, provided it is made to lie, by packing, in the natural position on the sternum, evincing but few signs of life, except the act of breathing, which is generally stertorous, now and then there is emitted a gurgling sound of gas and fluid regurgitated from the rumen.

The pulse, which at first is generally full, and more or less accelerated, becomes small, quick, and almost imperceptible; the breathing is more and more difficult; in some cases convulsions set in, whilst in others death occurs from a gradual sinking of the vital powers; the sphincters are paralyzed, the mouth open, and the breathing partially oral, with puffing of the cheeks at every expiratory act. The temperature is usually normal, but if much elevated, indicates some complication.

In some instances the symptoms of delirium are very violent; the animal struggles violently whilst down; works itself on its side in spite of all precautions; dashes its head wildly about; bellows, groans, and strains violently; very shortly the abdomen becomes tympanitic, and the breathing more laboured and difficult; there is eructation of foetid gases from the rumen, and death rapidly supervenes.

In other cases consciousness in some degree returns, the eyes become natural, the cow pricks its ears as if it once more could hear sounds, the secretion of milk returns, the body cools, and seems as if convalescence were in reality established. These flattering signs are, however, too often succeeded by a return of the worst symptoms, and it very frequently succumbs.

In some instances the bowels may be relaxed in the earlier stages; and this will greatly depend upon the previous treatment of the animal. This is, however, very soon succeeded by non-excretion both of fæces and urine. Constipation, or rather non-passage of the fæces, is always a marked sign, not that there is any actual dryness or costiveness, but that the muscular walls of the intestines are in a paralyzed condition, the peristaltic action being lost. The urine, generally pale in colour, and free from albumen, scantily secreted by the kidneys, is retained in the bladder, which has also lost its power of contractility.

## PROGNOSIS.

I consider it a good sign, if, after the animal has fallen, it is able to rise, although it fall again, and become perfectly comatose. This conclusion is a result of experience, but I can offer no explanation of it. If in the course of thirty to forty hours consciousness gradually returns, and the animal makes attempts, but not too violent, to rise, the bowels commence to act, the secretion of milk, and a desire for food return, a successful termination, at least of the apoplexy, may reasonably be expected. If, on the contrary, the sphincters relax, the coma becomes more and more profound, or the delirium continue, and if, in addition to these, the dropping of the jaw and oral breathing, which are always indicative of grave lesions, become manifest, a fatal issue is to be expected.

## POST MORTEM APPEARANCES.

The vessels of the body generally are filled with dark-coloured blood, the tissues are moist; the uterus is generally large and congested: this may, however, be unassociated with the disease, as it is the natural condition of the womb so soon after parturition. Extravasations of blood are found at the base of the brain, on the medulla, and cervical portion of the spinal cord. In some cases, however, actual rupture of the vessels has not occurred; great congestion of the vessels and the red points—*puncta vasculosa*—have been very marked when the brain has been cut with the knife, and in others no marked congestions have been seen, having in all probability disappeared *post mortem*, or perhaps immediately previous to the animal's death—congestion being generally an ephemeral condition; but in all instances there is an accumulation of fluid in the ventricles, and the brain substance itself is loaded with serosity.

The digestive organs, kidneys, liver, heart, present no special signs; the serous membranes are sometimes covered with petechial spots, the lungs are engorged as in all cases of death from paralysis of the respiratory apparatus. Thinking that perhaps the symptoms of intoxication seen in the disease were due to uræmic poisoning, I have very carefully examined the kidneys, but have never been able to detect any special lesions which

support that view. It is quite possible, however, that urea is superabundantly present in the circulation, but I do not think that it has any special connection with the origin of parturient apoplexy.

#### TREATMENT.

In the earlier stages, whether the animal is standing, or lying prostrate and in a state of coma, if the pulse be not excessively weak, and the heart's action almost fluttering, recourse may be had to venesection. Slow and deep breathing, with a tendency to stertor, add greatly to the necessity of immediate bleeding. The beneficial action of the withdrawal of blood is shown by the pulse becoming fuller, stronger, and better in tone. The opening into the jugular is to be a large one, in order that the blood may flow freely to relieve the congestion, to check, if possible, a further extravasation of blood or effusion of serum, and to divert its active flow into the head; but it must not be carried out so as to debilitate the heart's action. When the pulse becomes fuller and stronger, the bleeding is to be stopped; from three to five quarts will generally be sufficient.

The bleeding is for the purpose of removing pressure from the brain, and although the pulse may indicate stimulants rather than depletion, it will be found that as the blood flows the tone of the pulse will improve, for the weakness of the pulsation, the want of impulse, and debility of the heart's action, are results of brain pressure. If, however, the surface of the body be cold, the animal tympanitic, the heart's action fluttering, and the pulse almost undetectable, bleeding is calculated to do more harm than good, as the heart's action would now be further impaired, and the amount of arterial blood sent to the brain diminished; for it must be remembered that the cerebral congestion is now less due to an over-abundant supply of arterial blood than to pressure upon it by venous or capillary engorgement; that, in fact, the brain—engorged as it may appear—is in an anæmic condition in regard to its arterial supply; and when this is the case, paralysis of the heart is to be prevented, if possible, by the use of stimulants. It is the practice of many veterinarians, and of its success I can speak with confidence, to bleed

to relieve congestion, and administer a stimulant to increase the heart's action; to enable the heart, in fact, to send arterial blood into the brain by overcoming the resistance of the congestive venous pressure.

If the cow is already down when first seen by the practitioner, his first care must be to see that it is made to lie as near the natural position—on the sternum—as possible, and this he will do by having it packed up at the side by bundles of straw, or, what is better, sacks filled with straw, firmly wedged under the quarter and shoulder, the head at the same time being properly propped by the same means; and care being at all times taken that the cow is prevented from injuring its head by striking it against hard bodies. When this is done, venesection may be had recourse to.

In all cases, whether the pulse be small or full, a strong cathartic should be administered, consisting of sulphate of magnesia, with croton, ginger, or other aromatic, care being taken that the animal is not choked, as the act of deglutition is often performed with difficulty. I always try if the animal can swallow a little water from a bottle before administering any medicine. If the patient shows any difficulty in doing this, the medicine is to be given with the stomach pump. I am of opinion that the cathartic should not be repeated, having seen the repeated administration of even simple fluids cause tympanites and death. Cold water is to be applied to the head; the spine along its whole course stimulated with strong ammonia liniment, and the animal warmly, but not heavily, clothed. Every two or three hours it is to be turned and packed on the other side of the body; the mammary gland stripped and hand rubbed, and should there be signs of exhaustion or failure of the heart's action, a stimulant is to be administered; the best way of doing this is to give ammonia carbonate in a ball, which, after being oiled, is to be pushed by the hand as far as possible into the pharynx, when it will slowly gravitate into the rumen. If the tympanites is not relieved by the ammonia, the rumen is to be punctured by the trocar, and the cannula left in. Enemas of warm water are to be repeatedly injected into the rectum, the urine withdrawn, either by the finger introduced into the bladder, by the female catheter, or by introducing the nozzle of the ordinary injection syringe.

The pouring or pumping of large quantities of fluids, consisting of sundry quarts, or even gallons, of water, with treacle, ginger, &c., for the supposed purpose of causing purgation, is to be condemned; the fluid absorbed, whilst the excretory organs are not performing their functions, only adds to the general plethoric condition.

To prove the injurious consequences of this practice, as well as that arising from feeding the animal upon thin, sloppy food after parturition in order to prevent this disease, I will quote a parallel from human pathology.

Niemeyer says, under the head *Hypertrophy of the Heart*—“Such patients must beware of immoderate eating and drinking, in order to avoid the plethora which, although but transient, always follows the free use of food and drink. How often does the long-threatening apoplexy set in in the midst of the plethora which has developed after a long and hearty meal? In this connection, I may mention an act of folly which I have often seen practised by tavern-keepers and itinerant wine-dealers. The latter suppose that by a free use of water they can counteract the pernicious influences to which they expose themselves, although it is evident that the plethora arising after a full meal would only be increased by an immoderate addition of fluid.”

Schmidt, a most eminent Continental practitioner, being of opinion that the disease is due to the absorption of a poison generated in the udder, endeavoured either to prevent its formation or to destroy it before it became absorbed into the system.

*I have no hesitation in saying that this is one of the greatest, if not the greatest, discovery in veterinary medicine ever made, and the agriculturalists throughout the world owe Schmidt a deep debt of gratitude.*

His method of treatment is so successful that, what was looked upon as the scourge of the dairy farmer is now recognised as a disease which, in the vast majority of cases, terminates in a few hours in recovery. Since this discovery, the saving of life in dairy stock can only be reckoned in millions of pounds, and it is most extraordinary that the discoverer has reaped little or no reward.

The procedure is so seemingly simple that many dairymen

endeavour to follow it out themselves; but in their hands it frequently is unsuccessful—not, as they suppose, because it is worthless, but because they do not either understand or carry out asepsis in its strictest sense.

The principle of this treatment is to promote secretion of milk and destroy the poison in the udder; and this is done by, under perfect aseptic and antiseptic methods, injecting into each teat, by means of an air-pump or syringe, some antiseptic solution, and after the injection, the quarters of the udder must be well hand-rubbed and massaged.

Huish, of Red Lion Square, supplied me with a very good “milk-fever outfit,” which I have frequently used with marked success.

After the administration of the injection, and without any further medicinal treatment, nothing requires to be done but to make the animal comfortable, and remove the urine and fæces.

In most cases, signs of recovery are noticed within a few hours. In some cases, a second or even a third injection may be necessary.

Objection has been taken to this method of treatment, owing to the fact that cases of mammitis often occur as a result. If asepsis has been as perfect as is possible, these cases should be rare, and it is on this account that I urge that none but properly qualified persons should always be consulted in such matters.

When consciousness has returned, and the animal seems in a fair way of recovery, no medicines should be administered; but if at any time there are signs of exhaustion or sickness, small doses of spirits of nitrous ether and water may be administered, provided the animal is conscious and can swallow freely.

In some instances the appetite is inordinate immediately after consciousness has returned; the animal will greedily devour any hay or straw that may be about it. This must be prevented by muzzling it at once, or a fatal attack of indigestion with tympanites will be the invariable result. Small quantities of water or thin gruel may, however, be allowed, and all food given very sparingly until rumination has been fully re-established.

There are two sequelæ which often annoy the practitioner when he has congratulated himself upon a successful case, namely, congestion of the lungs, and paralysis of one or both hind extremities.

Congestion of the lungs appears about the third or fourth day. The cow, in the meantime, having perhaps been on its feet repeatedly, giving milk freely, &c., suddenly begins to breathe heavily and quickly, the pulse becomes oppressed and rapid, and the animal quickly sinks. In some instances the lung complication is due to inflammation of the bronchial tubes, excited by the incautious administration of fluids during the comatose stage; or the regurgitation of fluid from the stomachs into the partially paralyzed pharynx, which fluid may trickle down the trachea, owing to the loss of power of closing by the epiglottis; such fluids having passed into the trachea and lungs, and causing inflammation, which is manifested whilst the animal is otherwise recovering. An abundant secretion of mucus, with cough and mucous rales, indicate this, but undoubtedly congestion of the pulmonary system arises frequently, and independently of the irritation of foreign agents in the bronchial tubes, and is due to the malcondition of the blood, resulting from absorption of effete materials, and from debility of the heart's action. It is best treated with stimulants and fomentations to the sides, and the internal administration of antiseptics. It is, however, very fatal.

The other sequel—namely, paralysis of the hind extremities—very often baffles all attempts to overcome it, and is due to inflammation and red softening of the cord in the lumbar region. If the cow is not up about the second day after consciousness has returned and after it has commenced to improve, paralysis may be expected to be present. It is very true that some cows, out of sheer obstinacy, will refuse to rise upon their feet, although the practitioner knows well enough they can do so if they like.

Many a cow will do this until it sees some hateful object, such as a dog—and it is a common practice to introduce a dog into the byre—when her ladyship very soon takes to her feet. This is a harmless, and sometimes a very effectual practice. At other times, believing that paralysis is present,

and is to be overcome by severe counter-irritation, the veterinary surgeon has applied the actual cautery to the lumbar region. The touch of the hot iron has been enough; with a loud bellow and a dash, it has jumped to its feet, and, if not fastened, has made good use of its hitherto supposed paralyzed legs.

When paralysis is actually present, and if the animal is in good condition, it is better to destroy it—though most of the beef is fit only for second-class food—as too often the use of the limbs is never restored, more especially if it is a heavy, fat cow.

The treatment consists in the application of the actual cautery to the loins; blisters, purgatives, succeeded by iodide of potassium and diuretics, to excite the absorption of any exudation which may press upon the cord, and finally nuxvomica and sulphate of iron. In some instances one leg only is paralyzed, and the animal is able to rise with a little assistance. Many cows, however, resist any attempts to help them, and will struggle up if left alone; drag the paralyzed limb, “knuckle over” at the fetlock for a time, and finally recover.

I may state, in conclusion, that parturient apoplexy is a recurring disease, and that it is not safe to allow a cow to calve after it has once been down; and if the owner consults his own interests, he will milk it as long as possible, and then prepare it for the butcher.

I have heard of several cases of post-parturient paralysis in which dislocation of a hip-joint has been discovered *post mortem*.

A condition affecting the horse, commonly occurring in tropical countries, and even in England on very hot summer days, is the same as apoplexy from congestion, and commonly called heat apoplexy, heat-stroke, or sun-stroke. The symptoms are usually very acute and manifested suddenly. Horses in regular work and out the greater part of the day, such as tram horses, &c., are those usually affected, so much so, that in Bombay and Calcutta, and Southern States of America, before the introduction of electric traction, horses wore topees, or large thick pith hats, to act as sun-shades. Usually an animal suddenly staggers and then falls, and immediately



becomes comatose. The symptoms are alarming, and to the neophyte, the stertorous breathing, glassy eye, cold sweats, cold extremities, and complete immobility or spasmodic movements appear to indicate rapid dissolution. The pulse is slow and full, and respiration slow and laboured. Temperature at first low, but afterwards rises very high, and in the course of an hour may be  $106^{\circ}$  to  $107^{\circ}$  F. Prompt action is necessary, and the usual method is to apply cold water to the head—continued application by a hose-pipe if possible—friction to the limbs, and, on returning consciousness, a thorough rubbing until the animal is dry. This is followed by a purgative, aloes in solution and combined with calomel. Bleeding is not usually practised, but would appear to be indicated. The subsequent treatment must be directed as circumstances indicate. The liver generally appears to be congested, and requires stimulants such as ammonia chloride, soda sulphide, &c., general tonics, and an easily digestible diet of bran and scalded oats. In the majority of cases, if an animal recovers from the acute stage, convalescence is rapid, but occasionally animals are dull and unwell for some weeks. Subsequent complications are rare. There may be partial paralysis of a limb, or loss of co-ordination in a hind extremity; the tendency is to recovery, and the cause is probably pressure upon a nerve centre from some slight blood effusion. Treatment should be to endeavour to obtain absorption by pot. iodid. in combination with strychnia.

#### PARTURIENT ECLAMPSIA.

A nervous disease occurring in the lower animals shortly after parturition. It is most frequently seen in the bitch and sow, and only occasionally in the cow. When it occurs in the bitch, the usual message brought to the veterinary surgeon is that "So-and-So's bitch had pups a day or two ago, and has now gone mad."

The disease is due to absorption of toxins from the stomach and uterus, giving rise to severe nervous symptoms. It is usually ushered in by tremblings, twitchings, rigors, and convulsions, the patient being often delirious and unapproachable; it grinds its teeth, foams at the mouth, its eyeballs roll in their sockets, and the animal has a convulsive fit, and after

that has passed it may rise and be seemingly conscious, though weak ; but in other cases it may pass from fit to fit and then die, or it may become comatose, and either die in that condition or slowly recover.

I am of opinion that in many cases, as a result of the nervous shock due to parturition, there is great gastric derangement, the appetite is depraved, the patient may eat the foetal coverings, or may even eat its own young (which is not at all an uncommon thing), and as a result the toxins, which must be in abundance in such material, when taken into a disordered stomach, are highly likely to induce the symptoms as described ; or, in other cases, the cause may be due to the parent being deprived of its young, and as a result of inattention there is a great engorgement of the mammary gland, with the above sequelæ.

*Treatment.*—Prevent the animal from injuring itself. Apply cold water to the head. If necessary, give an emetic, followed by a dose of oil ; then give antiseptics, and thoroughly cleanse the womb. In cases due to mammary gland engorgement relieve the organs and endeavour to allay a probable mastitis.

## CHAPTER LIV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

(F.) ADVENTITIOUS SUBSTANCES IN THE BRAIN AND CRANIAL CAVITY—CAUSING, GENERALLY, SYMPTOMS OF CONDITIONS VARIOUSLY TERMED SLEEPY STAGGERS, IMMOBILITY, COMA, &c. &c.

#### TUMOURS.

TUMOURS are generally found in the choroid plexus. They are very commonly met with in both plexuses, sometimes larger in one than in the other; very often quite symmetrical in size and position. They grow slowly, and scarcely ever affect the health of the animal, or give any indication of their presence, until they have attained a size varying from a pigeon's to a hen's egg. They consist of a caseous material, mixed with a calcareous matter; they are in fact composed of an exudation which has undergone the caseous and calcareous degeneration. These calcareous particles are called brain sand—psammoma—and are never found *post mortem* presenting signs of recent origin.

I have found these psammomatous tumours in horses of various ages, but most commonly in old horses.

When they have attained the size above mentioned, they, in some instances, give rise to severe convulsive fits, a staggering gait and inability to perform work, whilst in others their presence is not indicated by any symptoms prior to a final stroke, simulating apoplexy, to which the animal rapidly succumbs. In those instances, when they occasion disturbance prior to the fatal attack, the symptoms vary somewhat in character; in one animal there will be unconsciousness, with elevation of the head and fore part of the body; in another—and this is rather a

frequent symptom—there will be a tendency to keep the head in a depressed position. In one case this depression of the head was very marked, but this did not happen until the animal—a mare—was worked or exercised; when kept quiet it appeared in the best of health in every way, but if exercised, no matter how slowly, depression of the head—the nose being almost brought to the ground—would always occur. This animal had, shortly before presenting these signs, recovered from an attack of farcy, affecting the cervical and facial absorbents. When I saw it I expressed an opinion that there was a tumour in or upon the brain, due to or connected with the previous farcy. I may mention that the cervical lymphatics were still enlarged, but showed no signs of recent inflammation. The probable age of these ventricular tumours is sometimes a question of importance as connected with the soundness of a horse prior to purchase; and it is well to bear this in mind, as an animal may die from an attack of cephalic disease, and upon a *post mortem* examination being made, these tumours may be found in the lateral ventricles. The clinical history of these points out that they are of slow growth; that they do not cause any disturbance until they have attained a certain size; they do not, at least in all the instances that I have met with or heard of, give rise to disturbance, increasing in severity as they slowly grow; but all at once, when they have attained the size of a small egg, signs of a severe cerebral disease occur, which ultimately proves fatal. Examination of the brain and tumours reveals—1<sup>st</sup>. The lateral ventricles nearly or completely filled by the growths; 2<sup>d</sup>. The tumours themselves firmly encapsuled in the folds of the choroid plexus; 3<sup>d</sup>. Absence of congestion, injection of the vessels, and other signs of recent inflammation; and 4<sup>th</sup>. A caseous and calcareous change of the interior of the tumours.

*Thickening of the dura mater.*—Thickening and induration of the dura mater consequent upon a slow chronic inflammation is of very rare occurrence. I have one case on record, however, in which the dura mater attained a thickness varying from one inch at the base to several inches at the anterior part of the cranium, causing absorption of the descending (orbital) plates of the frontal bones and wings of the ethmoid, and filling the frontal sinuses. The horse in which this was discovered had presented signs of brain disease, coma, immobility, partial

paralysis, amaurosis, and paralysis of the muscles of mastication, for a considerable period before its death. The *post mortem* examination revealed that the bones above mentioned had been absorbed, the frontal sinuses filled with a white mass resembling brain, but which proved to be thickening of the dura mater; the brain compressed, very solid in consistence; the lateral ventricles almost obliterated by approximation of their sides and roof, and the brain generally anæmic. There was no previous history to this case. It is very probable, however, that this condition was a result of an injury, such as a blow upon the head, causing perhaps partial detachment of the dura mater without fracture of the bones, and consequent chronic inflammation.

In another case there was enlargement of the lachrymal gland causing absorption of the bones of the orbit, and death by pressure on the brain. The cranial bones as well as the brain are now in my possession, and were obtained from an old horse, which had presented no signs of disease, except a slight difficulty in masticating its food for a few days prior to death, and at first was supposed to be another specimen of induration of the dura mater: but further examination proved it to be due to enlargement of the lachrymal gland. In addition to penetrating the cranium, the tumour partly filled the frontal sinus on the same side, causing partial absorption of the orbital process of the frontal bone; but the most remarkable circumstance in connection with the progress of the case was the fact that at no time was there any bulging or undue prominence of the eye; a condition that one would have supposed would have been a more likely one to occur than absorption of the osseous walls of the orbit.

The tumour measured several inches, was lobulated in character, and microscopically was found to consist of numerous cells imbedded in a few very delicate fibres. The tumour presented no signs of retrograde change, and partook of the character of lymphadenoma in many particulars.

#### TUBERCULAR DEPOSITS IN THE MENINGES OF THE BRAIN.

Tubercular meningitis—a disease not rarely met with in the human being, more especially in the young—is a very rare affection in the lower animals. I have seen it in calves about three months old.

The symptoms were those of nervous derangement, partial paralysis, squinting, contraction of the pupils, fits of convulsions succeeded by coma, loss of motor power, amaurosis, and death. In some cases there will be indigestion, purging, the fæces being white, and consisting of the milk the animal was fed upon, loss of flesh, hardness of the belly, and emaciation. In others the digestion may be good and the body fairly nourished.

In addition to small tubercular deposits, scattered here and there on the membranes—caseous and calcareous, scrofulous tumours were found in the peritoneum, in the mesenteric glands, and in the pleura. Very probably the condition termed hydrocephalus, or dropsy of the brain, is due to the development of tubercle during foetal life.

#### EXOSTOSES

Are met with in horned cattle. These consist of enamel-like growths of a globular nature, sometimes convoluted, and attached by a pedicle. They dip, along with the dura mater, into the convolutions of the brain, and seem to be due to ossification of the dura mater.

Mr. Gamgee reports a tumour having the appearances of an ossified brain, which is in the Milan Museum; and states that these growths sometimes attain the size of an ox's brain, without inducing any apparent disorder until the animal's sudden death.

I have seen in the horse tumours having a resemblance to dentine invading the temporal bones, both externally and internally; and these, like the bony tumours of cattle, have induced no marked symptoms during life.

#### HYPERTROPHY AND ATROPHY OF THE BRAIN

Are conditions unknown in the lower animals. In my work on the *Principles and Practice of Veterinary Surgery*, page 182, a case is described of apparent hypertrophy of the brain; but in reality the apparent enlargement was due to development of fibrous tissue in the brain, which of itself was not enlarged. In the case of thickening of the dura mater mentioned at page 504, the brain was atrophied by compression owing to the thickening of the dura mater.

## MELANOSIS OF THE BRAIN AND MENINGES.

Small, black, melanotic nodules, varying in size from a pea to that of a bean, were found in the brain and on its meninges in an aged grey stallion which had suffered for a number of years from "stringhalt." Not only were they found in the cranial cavity, but along the course of the spinal cord, being particularly numerous in the lumbar region.

## ENLARGEMENT OF THE PINEAL BODY.

This body is sometimes found enlarged, and sometimes converted into a cyst, containing a whitish viscid fluid. The pituitary body may also be found in a similar condition. These appearances are met with in old subjects on the dissecting table. There are no appearances which indicate their presence during life.

*Parasites in the Brain.*—(See *Parasitic Diseases*.)

## SOFTENING OF THE BRAIN.

I have already stated that softening of the brain occurs as a sequel of epizootic cellulitis, and that it is due to plugging of the cerebral arterial branches.

All the conditions mentioned in this chapter may give rise to what is termed *immobilité* by French veterinarians—coma somnolentum by writers on human medicine, and sleepy staggers by old writers on farriery; a manifestation or expression of brain disease characterised by general dulness, a tendency to fall asleep when allowed to stand quiet for a while, tripping or falling, staggering gait, slow pulse, and deep respiration. An animal suffering from this will often fall asleep with its mouth full of food. It is, however, easily aroused, and if this be done suddenly, will manifest fright, trembling, and a disposition to delirium.

For the relief of these symptoms an occasional purgative ought to be administered, the animal carefully fed, and worked gently, so long as it remains serviceable, after which it should be humanely destroyed, as it is useless to endeavour to cure what is organically changed. Hence all irritating materials in the form of blisters, setons, &c., should be avoided.

## CHAPTER LV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (G.) DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

INFLAMMATION of the spinal cord and its meninges is described as two separate diseases by human pathologists, and is termed myelitis, when attacking the substance of the cord; rachidian arachnitis and spinal meningitis, when involving the membranes only. I think that in the lower animals the distinction is unnecessary. I shall therefore describe the disease under the generic term.

#### SPINITIS.

Idiopathic inflammation of the spinal cord and its membranes presents itself in the horse in two forms, namely, acute and chronic.

The symptoms of acute idiopathic spinitis are not always alike. In some instances the paralysis, which results in every case, may be manifested suddenly, and with little or no previous premonitory signs of ill health, the animal losing all power of movement over all parts posterior to the seat of inflammation; whilst in others, symptoms of cramp or spasm, great restlessness, and a high degree of fever may for some time precede the paralysis.

The following case will illustrate this form of attack:—A grey cart gelding, six years old, in previous good condition, and at regular work, was observed to perspire profusely, to breathe quickly, and to express signs of great restlessness; the hind feet were alternately lifted from the ground with great



violence, the great muscles of the thighs were violently cramped; and such was the degree of pain that it became almost unmanageable. It was led home and placed in a loose box, where it lay or almost fell down. For a while it seemed relieved, but after a time commenced to struggle violently, and at last managed to regain the standing posture. The cramp of the limbs returned; after an interval it again lay down, and when first seen by me it was struggling violently, breathing heavily, and groaning with pain; the pulse about ninety, and full; mucous membranes injected, and the body bedewed with sweat. It again struggled to its feet, passed some rather high-coloured urine; remained quiescent for a short period, and then the great struggle again commenced. It was placed in slings, into which it threw its weight, and gave a sigh of relief; after an interval of about fifteen minutes it commenced to struggle violently, its hind legs were dreadfully cramped, and we were obliged to lower it down on to its bedding, when it again seemed to receive relief. It was bled and a purgative with belladonna administered, the loins and back were fomented with hot water, mustard applied, and afterwards covered with a sheep-skin. In a while, however, it commenced to struggle afresh, and we were compelled to raise it by pulleys and slings. For two days and two nights it thus remained, now and then free from pain, then in dreadful agony; it seemed that all movement hurt it dreadfully, every jerk and start causing it to groan and to jump as it were in agony. At first the muscular convulsions were confined to the posterior extremities, but the fore limbs became after a time affected. The poor horse seemed to dread moving its limbs, but a sudden start would ensue, and all its limbs would move convulsively and with great force. We were compelled alternately to sling and let it down, for in no posture could it obtain ease for more than a few minutes at a time. On the third morning the spasms left it, the bowels responded to the purgative, the respirations became slower, but the pulse was feeble, thready, and greatly accelerated, beating about 130 per minute. It could now stand for some hours, but if made to move it did so with great difficulty, dragged its hind limbs, staggered, and was inclined to knuckle at the fetlocks; the off fore limb was also partially paralyzed, and was moved with difficulty. When last seen by me it was standing with its head over

the box door, and looking pretty cheerful, had drank some gruel and eaten a little hay, and I was in hopes it might rally; but shortly afterwards it fell completely paralyzed, lost both motor power and sensitiveness; there was no struggling now, it lay passively on its side, looked quite conscious, and died without a struggle. A *post mortem* examination revealed the spinal cord and meninges highly congested, and the subarachnoid space filled with serosity. The dorsal, lumbar, gluteal, and the muscles of the thighs, as well as the serrati magni, levatores humeri, triceps extensor brachii, and the other muscles which had been violently cramped during life, presented a dark, congested appearance.

The spinal congestion was most marked in the dorso-lumbar region, and both roots of the spinal nerves, and the nerves themselves for some distance, presented the same appearance. The arachnoid, pia mater, and dura mater were intensely red; the substance of the cord looked reddish, and on cutting through it black blood exuded abundantly from its centre.

The other form of spinitis is that manifested by sudden loss of power, without previous irregular muscular action, and may be appropriately termed inflammatory paralysis; for, in addition to the paralysis, there is fever, constipation of the bowels, diminished secretion of urine, and other signs of constitutional disturbance. This form is apt to be confounded with fracture of the vertebræ, more especially of the lumbar vertebræ, in which generally the paralysis is complete immediately after an accident. Whereas, when one of the dorsal vertebræ is fractured, displacement may not immediately occur, displacement and paralysis occurring perhaps in a few hours after the accident.

The history of the case will have to be taken into consideration in arriving at a diagnosis. Broken back generally succeeds a traceable accident, acute spinitis without any appreciable cause.

The first form of spinal inflammation is apt to be confounded with azoturia. The dark colour of the urine which characterises that disease is absent in spinal inflammation, and the spasm, which in azoturia is mostly confined to the gluteals, affects various muscles, and is of a more clonic or alternating kind.

## CHRONIC SPINITIS.

This again presents itself in two forms, namely, that associated with tonic spasm of the voluntary muscles, and that associated with paralysis of particular muscles.

Chronic spinitis associated with exalted muscular contractility is in some districts termed the "cords," from the fact that the superficial muscles have a prominent or corded appearance.

The symptoms are at first obscure. There is some degree of stiffness about the spine, the animal turns with difficulty, and often groans when made to do so suddenly. Some particular muscle is now seen to stand out from its fellows, and present a permanent rigidity and hardness. Very often the muscles of the shoulders or the levatores humeri and cervical muscles are first affected; but gradually the whole body becomes more or less stiff, the limbs are flexed with difficulty, the nose elevated, and the animal seems as if affected with chronic tetanus; the facial muscles, however, remain normal, and the animal continues to feed well. After the lapse of some weeks, the hind limbs become feeble; there is knuckling over at the fetlocks, crossing of the feet, staggering gait; the animal seldom lies down, and rises with great difficulty, and eventually becomes paralyzed, and has to be destroyed.

In the other form the first symptoms are, staggering gait, weakness of the hind legs, crossing of the feet, and a gradually increasing loss of motor power, until finally the animal becomes powerless, unable to rise from the recumbent posture, and has to be destroyed. In none of these cases—acute or chronic—do we find that the sphincters lose their power of contractility, nor is common sensibility lost. Indeed, in the acute form, I have often thought there was hyperæsthesia, and that the animal dreaded the touch of its attendant. These conditions indicate that the meninges are the principal seat of the inflammation.

*The post mortem.*—The spinal cord is red, injected, and softened (red softening), the meninges thickened, the arachnoid covered with an exudate, which in some places joins its opposed surfaces, or is filled by a semi-purulent or reddish serosity. In one case, which died during very cold weather, the fluid, being frozen, after death presented a very beautiful appearance.

Idiopathic spinitis differs from that arising from external

injuries or disease of the bones, in which the dura mater is inflamed on its adherent surface; the areolar tissue uniting it to the spinal canal is loaded with venous blood, or sometimes broken down, and the dura matter separated from its attachments.

The causes of idiopathic spinal inflammation are very obscure. Very probably it is often due to rheumatic inflammation localized in the membranes primarily, the cord itself becoming secondarily affected. In *Principles and Practice of Veterinary Surgery*, page 248, I have pointed out that hereditary tendency is a cause of spinal affections.

#### TREATMENT.

The acute forms are to be treated antiphlogistically; blood may be withdrawn and purgatives administered; depletives must not, however, be pushed too far, as acute spinal diseases have a lowering or debilitating effect upon the system. The purgative may be combined with belladonna, which will to some extent relieve pain and spasm, as well as overcome the congestion of the spinal vessels. Hot fomentations to the back and loins are indicated in the earlier stages, and blisters, or even setons, should there be but a partial recovery. It must be remembered that a continuance of the symptoms, after the acute stage has passed off, depends upon some degree of effusion or exudation, and that means must be taken to promote absorption. On this account iodide of potassium with diuretics is indicated. Ergot of rye (*Secale cornutum*) in combination with the iodide of potassium is well spoken of by physicians, and may be tried in the lower animals.

In all cases great care must be taken that the urinary bladder be regularly emptied; and if micturition is not naturally and easily performed, the catheter must be used at least thrice a day. If the animal is recumbent, this should never be neglected. The attendants must also be directed to turn the patient every now and then, see that enemas are regularly administered, and that the bed is kept clean and dry.

## CHAPTER LVI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

##### (H.) PARALYSIS.

PALSY or paralysis may be seen in various forms rather as a symptom of a lesion than as a disease itself, the term *palsy* or *paralysis* being commonly restricted to that form where motion is lost, while the term *anæsthesia* implies a palsy of the nerves of sensation.

In order to understand the various lesions which may cause paralytic symptoms, it is advisable that a glance at the physiology of the spinal cord should now be undertaken.

1. The spinal nerves have two roots; and Sir Charles Bell proved that the anterior (inferior in the lower animals) conduct the power of motion, whilst the posterior (superior) are devoted to sensation only. At one time it was thought that if the corresponding columns of the cord were cut or injured, loss of sensation only would result when the superior columns were cut, and loss of motion if the inferior were cut across; but such is not actually the case, for it has been demonstrated that if the posterior columns are cut across, the result is not numbness and insensibility, but hyperæsthesia and loss of co-ordinating power in the parts posterior to the section, with some local pain, due not to any sensitiveness in the columns themselves, but to the cut having traversed through the posterior roots of the nerves. The same results are obtained if the restiform bodies and small superior pyramids are divided, and as these parts are connected with the cerebellum, and the superior columns of the cord, the deduction is, that the channel which

conducts the action of the cerebellum upon the body is composed of the restiform bodies and small posterior pyramids in the anterior part, and of the superior columns of the cord in the posterior part of its course.

Section of the inferior columns, if made immediately behind the medulla oblongata, is not followed by any very obvious loss of paralysis or loss of sensation, unless the cut be extended to the lateral column, when loss of motion is the result, as if the columns were divided behind this point. It is therefore apparent that in the anterior part of their course the inferior columns have not an intimate connection with the corresponding roots of the spinal nerves. Posterior to this, however, they have a close connection with the inferior roots of the spinal nerves and with voluntary motion. They have also some connection with sensibility, as a certain degree of numbness is produced by injuries which give rise to loss of motion.

*Inferior pyramids.*—Section of one of these pyramids in any part of its course is followed by immediate loss of voluntary movements in the muscles below the cut on the opposite side of the body; but sensation is not affected in any appreciable manner.

*Lateral columns.*—In the cervical region, for a short distance behind the point at which the inferior pyramids of the medulla oblongata decussate, the lateral columns of the spinal cord are directly concerned in conveying the stimulus of the will to the muscles on the same side of the body, for when they are cut across paralysis of the muscles of the same side occurs.

In the lower part of the neck, and regions posterior to this, the same results are not seen; for when they are divided in these regions, some trifling paralysis ensues, with some degree of anæsthesia and loss of co-ordination.

*Olivary bodies.*—When one of these is divided, a persistent spasm of many muscles of the same side is the result.

*Grey substance of the cord.*—Dr. Brown Sequard says that the grey substance of the cord is an important conductor of sensory and motor impressions. Paralysis, without loss of sensation on the same side of the body, are the results of cutting across one lateral half of the grey substance of the cord. Anæsthesia on both sides of the body, paralysis of neither side, are the strange results of making a longitudinal section midway between the two lateral halves.

The superior half of the white matter may be divided at one point, and the inferior half at another a little anteriorly, so that all the white fibres shall be divided transversely by the one cut or the other, without any material continuity of the cord or damage to the grey matter; and when this has been done, irritation of the sensory nerves connected with the parts below the section excites the sensation of pain as strongly as ever. Hence it follows that the impulses which excite pain reach the brain through the grey matter, and so long as a small portion of the grey matter remains intact, these sensations are transmitted.

If one-half of the cord be cut through transversely down to its very middle, so as to interrupt all continuity of both white and grey matter, irritation of the skin of the same side will give rise to as much pain as if the cord were not cut, but all voluntary power will be lost in the muscles of that side below the section. It thus follows that the channels which convey sensory impulses must cross over from the side of the cord which they enter to the opposite side, and that the motor influences sent down from the brain must travel along that side of the cord by which they pass out.

There is increased temperature and sensibility on the side in which sensation is preserved, and diminished temperature on the side in which sensation is lost, especially if the section is made near the medulla oblongata.

It would seem that the injury acts upon the vasa-motor nerves contained in the cord, as well as upon the motor and sensory nerves (it may be here stated that the vasa-motor fibres do not arise from the sympathetic ganglia, but simply pass through them on their way from the spinal cord to the upper dorsal region), causing paralysis of the vasa-motor nerves on the side in which there is increased temperature and sensibility, and irritation of the vasa-motor nerves on the side in which there is diminished temperature and anæsthesia; for the experiments of Dr. Brown Sequard, Claude Bernard, and others have proved that when the cervical sympathetic is paralyzed by dividing it, a state of congestion, of which the most conspicuous signs are a blood-shot state of the conjunctiva and lining membrane of the ear and nostril, with a contracted pupil, and increased temperature, is at once set up on the same side of the head, and also

that when the end of the divided nerve is irritated, the immediate result is dilatation of the pupil, with an immediate cooling and blanching of the parts which were blood-shot and warm.

Injuries to the cervical portions of the spinal cord are followed by a remarkable elevation of the animal heat. In one case of injury in a man where there was a forcible separation of the fifth and sixth cervical vertebrae, with extravasation of blood into the theca vertebralis and laceration of the lower part of the spinal cord, although the breathing was performed by the diaphragm only and very imperfectly, the pulse weak and countenance livid, the thermometer indicated 111° F.—(HUXLEY, BROWN SEQUARD, CLAUDE BERNARD, Dr. RATCLIFFE.)

Such, then, are a few facts which serve as indications of the seat of spinal lesions.

In all cases of disease or injury to the cord the paralysis occurs in the parts to which all the nerves originating posteriorly to the seat of the lesion are distributed. If the injury or disease be in the sacrum the tail alone is paralyzed; if in the lumbar region the hind legs are paralyzed; and if the disease or injury be very great the sphincters of the anus and bladder may be paralyzed. If the injury be higher up, in addition to loss of voluntary power in the hind limbs, the abdominal muscles will be paralyzed, and the expiratory movements performed with difficulty.

If the disease be low down in the cervical region, the muscles of the fore limbs, as well as those of respiration, will be paralyzed, and both expiratory and inspiratory movements will be difficult; and if the injury be above the middle of the neck, or anterior to the fourth pair of spinal nerves, death will at once result from paralysis of the diaphragm, the serrati magni, scaleni, intercostales, and other muscles of inspiration.

Paralysis as seen in the lower animals may be divided into—1st. Paraplegia; 2d. Hemiplegia; and 3d. Paralysis originating in injury to a motor nerve, as paralysis of the lips, described at page 496 of *Principles and Practice of Veterinary Surgery*.

#### PARAPLEGIA,

Or *Paralysis affecting a portion of the body transversely*.—Of this kind three forms are met with in veterinary practice, namely



—1st. Centric, or that arising from disease of the spinal cord or its membranes. Symptoms simulating paralysis may occur from occlusion of the iliac arteries.—(See *Veterinary Surgery*.) 2d. Eccentric or reflex paraplegia; and 3d. Paralysis due to blood poisoning.

Of centric paraplegia but little need be said; it is symptomatic of conditions of the spinal cord and its membranes already described, or it may result from injuries to the cord arising from violence, fractures, &c.

#### REFLEX PARAPLEGIA.

In horses, cattle, and dogs, spinal paralysis is met with in which no disease of the cord or its meninges can be detected *post mortem* in those which die, and which, in many cases, is quite ephemeral, passing away when its cause is removed.

The causes of reflex paralysis as seen in the horse are those originating in indigestion, constipation of the bowels, and, in the mare, uterine irritation.

Paralysis arising from indigestion is occasionally witnessed during attacks of colic, the loss of power coming on rather slowly; the animal is at first seen to show weakness of one hind limb, with knuckling over at the fetlock, and difficulty of moving it, then the opposite limb may become affected; and if the loss of motor power be great, the animal falls upon its haunches, and is unable to rise. If it be a docile animal and will lie quietly, the paralysis may disappear in a few hours, or leave only such traces as weakness of the limbs and staggering gait, which may take some days to pass away.

In mares, during the period of oestrus, I have repeatedly seen cases of paraplegia, generally preceded by some irregular muscular action; persistent tonic spasm of the muscles of the hind quarters, continual kicking with the hind feet, with great injection of the vulva, succeeded by loss of motor power, and even death.—(See *Hysteria*.) In dogs, paralysis, even affecting all parts of the body except the head, is not an infrequent result of the irritation of teething, rich foods, constipation, and want of exercise. In horned cattle paraplegia is not an infrequent symptom of indigestion, arising from impaction of the rumen, and from post-parturient uterine irritation. I have already

mentioned a case of general paralysis succeeded by congestion of the brain and death in a goat, arising from grief at the loss of her kids; this may justly be called emotional paralysis.

The differences existing between reflex paralysis and that from disease of the cord is found in the circumstance that recovery is not at all an infrequent result, and that when death occurs either from the gravity of the primary disease, restlessness of the animal, or other circumstances, no spinal *post mortem* lesions are discoverable.

It is supposed by Dr. Brown Sequard that reflex paraplegia is not due to spinal congestion, but to a condition diametrically opposed. He believes that a state of irritation, commencing eccentrically, is propagated along the vasa-motor nerves, of which the result is, primarily, contraction of blood-vessels in, and, secondarily, exclusion of the due amount of blood from, one or more of the three parts following—the spinal cord, the nerves proceeding to or coming from the cord, the muscles, and that the proper activity of the nervous tissue is starved into paralysis from want of blood. This view is founded by Dr. Brown Sequard upon the fact that a state of irritation in the vasa-motor nerves may proceed from a distant point, and produce contraction of the vessels, and upon the fact that traces of organic disease are wanting after death in many cases of reflex paraplegia.

The following are among the causes of reflex paraplegia in the human being:—Irritation of the urethra; inflammation of the bladder; diseases of the prostate and kidneys; enteritis; the presence of worms in the intestines; dysentery; diphtheria; diseases of the lungs and pleura; the irritation of teething; irritation of the cutaneous nerves generally following cold and wet; and diseases of the knee joint.

Four different conditions of the muscles are observed in paralysis in the human being, some of which are also observable in the lower animals:—(1.) A condition little different from that of health, but less firm, less excitable by the galvanic stimulus, when the paralyzing lesion is not of an irritative kind. (2.) Complete relaxation of the muscles, characterised by imperfect nourishment and rapid wasting—so rapid that in a few days the size of a limb experiences a marked diminution. Such muscles scarcely, if at all, respond to the galvanic stimulus. (3.) Contraction of the muscles, with rigidity and wasting (the

flexors being always more rigid than the extensors), a condition which is due to chronic shortening of the muscles themselves, and generally associated with muscular atrophy. (4.) Nutrition not impaired, constant firmness and rigidity, incomplete paralysis, increased susceptibility to galvanic stimulus.

In the treatment of reflex paraplegia the removal of the eccentric cause must always be the primary object of the practitioner. In the horse, if there be colicky pains and constipation, opium and purgatives, succeeded by belladonna, and an alkaline carbonate; the soda bicarbonate in all cases arising from indigestion. If congestion of the cord is suspected, belladonna or the ergot of rye are indicated. Generally, however, it is found that in the lower animals reflex paralysis is ephemeral, and passes away when its cause is removed.

*In the cow* the stomach and bowels are to be unloaded by strong cathartics. Sulphate of magnesia, with croton, if there be no actual inflammation of the stomachs; oleaginous purgatives, with opium, belladonna, or hyoscyamus, if inflammation is suspected.

*In the dog* reflex paralysis is often accompanied by great irritation of the stomach, vomiting, and intestinal pains. Castor oil and hydrocyanic acid are the most useful agents in affording relief. If the stomach be too irritable to retain the oil, the hydrocyanic acid is to be given alone, or in combination with carbonate of soda, until undue irritability is allayed, then the aperient is to be administered. Iodide of potassium and the soda carbonate agree better, and are more useful in the dog than all other remedies after the aperient has operated. If the disease be due to decayed teeth or the irritation of teething, relief must be afforded by extraction of the teeth or lancing of the gums. In all animals cathartics, enemas, fomentations to the loins, hot baths for the dog, and emptying of the bladder, where possible, are not to be lost sight of. If paralysis continue for a time, nux vomica and ferruginous tonics may be prescribed advantageously, and the loins may be blistered, and galvanism applied.

#### HEMIPLEGIA.

Hemiplegia is that form of paralysis in which one lateral half of the body is affected. It is a very rare form of disease in the lower animals. I have, however, seen it in the horse,

and Mr. Mahony, V.S., Templemore, in a letter to me, December 1878, describes a well marked case. I have also seen it in the cow and in the dog.

M. Girard, Jun., has left a case on record, quoted by Mr. Percivall, which bears out many points in the physiology of the cord already referred to. He says—"The sensibility of the left—the affected—side proved extremely acute. The lips and alæ of the nose were drawn to the right side, the contrary to that to which the head and neck turned. The occlusion of the nostrils was such that the air made a blowing noise in its passage through them. The left ear was palsied, and the tongue slightly distorted. The lips and nostrils retained their sensibility, though in a diminished degree to what it was on the unaffected side. When oats were laid before the horse, it seized them with the right side of the mouth, the left remaining motionless. It experienced a great difficulty in mastication, and succeeded only in swallowing a part of its food, the remainder staying behind, lodged between the cheek and molar teeth. It could manage to pick up its oats from a plain surface, and when presented to it in a trough, it plunged its muzzle into the middle of them, opening wide its mouth. It could drink but slowly, and with difficulty, and only by thrusting its mouth deeply into the water. The nostril of the affected side perceived scents. It could walk, but could hardly sustain itself after but a short exercise. If attempted to be turned to the left side, this instability became still more manifest. Pressure upon the vertebral column from the head to the tail seemed to give great pain. The respiration, although sonorous, was regular. On the fourth day, the animal, unable any longer to stand, sank down, and after several turns and ineffectual struggles to rise, rolled over and lay upon its right side. Its bowels were relieved by manual operation, its bladder with the catheter; though even after this it passed its dung, but could not posture itself to pass its urine. Its pulse, like respiration, remained undisturbed. It died on the seventh day."

In the case of the cow the attack was of an acute kind. The animal was grazing in a field with a lot of others, and was left quite well at milking time in the morning. The field being some distance from the house, it was not seen again before evening. It was then found prostrate on the ground, lying upon its left side, and was unable to rise.

When I saw it I observed the following symptoms:—The left ear was pendulous, left eyelid drooping and closed, the eye squinted outwards. The left lip hung down, the angle of the mouth was lower than on the opposite side, the muscles were loose and flaccid, and the lips drawn to the right side. The tongue protruded, and when put into the mouth was drawn to the right side. The neck was twisted and the head drawn to the left side. Deglutition was imperfect, but the breathing was not appreciably affected. The superficial blood-vessels of the right side were engorged with blood, and stood out prominently all over the trunk and neck. The same side was warm, whilst the opposite side was cold, and the hair was “pen-feathered.”

In endeavouring to place the animal on a hurdle for the purpose of removing it to a shed, it was observed that it rolled over from the left to the right side, on which side only could it be made to lie. The bowels were constipated, and the belly tympanitic, the sphincter ani was rather relaxed, the vulva flaccid, and vagina protruding. The animal was quite conscious, but inclined to somnolency. It was bled, a purgative administered, and the back was fomented and dressed with strong ammonia liniment. On the following morning it was able to rise, but both the legs of the right side remained partially paralyzed for some weeks. The affection of the face, however, passed away during the first night, and it was able to partake of food on the following morning.

This was a case of hemiplegia due to some ephemeral disease of one side—the left—of the brain; and from the fact of the outward squinting, amaurosis, and drooping of the upper eyelid, the conclusion is, the functions of the third nerve being interfered with, that the brain disease—ephemeral as it was—involved the posterior part of the left crus cerebri. It must be remembered that, in all cases of disease affecting one side of the brain, the paralysis is on the opposite side of the neck and trunk, but on the same side of the face. It was remarkable however, in this case, that although the loss of motor power was complete, sensibility remained intact.

Paralysis from blood poisoning is witnessed in azoturia, lead poisoning, canine distemper, and grass staggers. In the human being, as well as in horses, cattle, and birds, a form of paralysis arises from the use of the vetch known as the *Lathyrus sativus*.

—(See Lathyrism, page 583.) The canine distemper poison leads in many instances to softening of the cord, in which its substance is broken down and softened almost to a pulp. In recent cases the softened cord is red or yellowish-red in appearance, due to some degree of accompanying congestion. In the more chronic cases, or those which have lived for a longer period, the red appearance is absent. White softening may, however, occur quite independently of the red or inflammatory form, and is due to cedema or effusion of serum. I have repeatedly seen not only softening but apoplexy of the cord occur as a sequel to canine distemper, and in which the neurilemma of the roots of the nerves were filled by a clear fluid material, and quite devoid of true nervous matter. In softening, the grey matter of the cord is always more affected than the white. I have never seen the converse condition of the cord, namely, hypertrophy, nor induration of its substance. The treatment is the same as that for paraplegia, and in the case of a valuable animal galvanism should be tried for a prolonged period. Young sheep grazed on newly limed land are often affected with spinal paralysis.

#### LOUPING-ILL, OR TREMBLING.<sup>1</sup>

LOUPING-ILL.—The term “loup-ill,” supposed to be derived from *hloupa*, an old Scandinavian term for “a staggering gait,” as applied to the disease, is general in some parts of Scotland, whilst in others it is known by the term “trembling.” It seems to prevail more in the Silurian hill districts of Scotland, and extends from the Hebrides to the southern extremity of Dumfriesshire, being equally prevalent in the district of Langholm as in the isle of Skye. There is this difference, however, namely, in the southern parts it rarely occurs to any extent but during the early summer months, commencing about the middle of April, and disappearing before the end of June; whereas in Skye there are two annual outbreaks,—one in early summer, and another in autumn, commencing at the end of August, and terminating in a month or six weeks.

It is stated that it affects, but to a slight extent, cattle, horses, and pigs. I have seen well-marked cases in horned cattle, but never in horses and swine.

*Symptoms.*—The symptoms, as observed during my investigations in 1881 and 1882, were as follows:—

<sup>1</sup> Condensed from Reports to the Highland and Agricultural Society of Scotland, 1882-97.

No. 1.—A three-year-old ewe was seized on the 18th May, just before giving birth to a dead lamb. When seen on the 19th she was standing with head erect and brilliant eye, the upper muscles of the neck spasmodically contracted, causing the nose to be protruded as in tetanus. When made to move, she did so very stiffly and automatically, the limbs being lifted very high, and the feet planted on the ground in a peculiar jerky manner, like a horse with stringhalt. There were tonic or persistent spasms of the muscles of the loins, back, and neck, and clonic or intermitting and irregular spasms of the other muscles, particularly those of the limbs; the breathing was very jerky, quick, and irregular, being from fifty to sixty per minute when quiet; pulse 102, and temperature 105.5° F.

On being excited the ewe fell prostrate on the belly and chest, and lay with head and neck extended; on being raised, walked away trembling violently, the trembling being due to alternate contraction and relaxation of the muscles.

On examining the skin three kinds of ticks were found, but they were not very numerous.

She was killed, and on making a *post mortem* examination of this ewe, nothing very special was discovered; the flesh was pale, and the carcass thin, but there seemed to be no abnormality of the cord or spinal canal, beyond a slight increase of the spinal fluid. There was no jelly-like exudate on the cord, nor any congestion of the cord, or of the membranes.

No. 2.—Another ewe was recovering. She had trembled for a day, then became paralyzed, and went down and lay three days, but was then up and walking about, but in an unsteady semi-paralyzed manner.

No. 3.—Ewe hogg; had been ill four days; was not observed to tremble, but found down, paralyzed. Was in good condition; in fact almost fat. Lay quietly on the right side; eyes open, no dilatation of the pupils, nor any squint. When suddenly approached, or otherwise disturbed, was very frightened, and struggled violently. When hind leg was lifted up and allowed to fall there was no attempt at motion, but when the fore leg was lifted in the same way there was a to-and-fro motion before reaching the ground.

When raised, and held up by hand, it could move its fore limbs pretty freely, and could stand and bear weight upon them;

but the hind ones remained extended backwards, completely paralyzed, and the muscles of the hind quarter were clonically convulsed.

Sensibility was diminished in the hind limbs, but there seemed to be some degree of hyperaesthesia in the fore ones, as these moved very rapidly and violently when pricked. The sensibility of the trunk seemed to be normal.

The temperature was 105·5° F., pulse 106, and respirations, when not disturbed, 30, but when excited the breathing became very rapid, jerky, and convulsive. The *post mortem* examination revealed nothing very particular in the spinal canal, brain, nor in any other organ. The flesh was not at all pale or anæmic, as in the majority of the cases examined, and fat was abundant and very healthy looking.

During 1881 about three score of sheep and lambs were examined after death, and about one-third of these were considered to have had "loup-ill," whilst others were found to have had—(1.) "Joint-ill" (suppurative arthritis); (2.) Anæmia, from pure starvation; (3.) Impaction of the stomach with wool in young lambs; (4.) Abscesses in various parts.

In one lamb, sent in from Dumfriesshire in 1882, there was a downward squint in both eyes even when in repose, but in others there was no squinting unless under excitement, when, as in the first case described, there was often a convulsive motion in both eyes.

We were informed that many sheep seemingly in good health often made a jump and fell down, some dead, some completely paralyzed, when suddenly frightened or approached by a dog, and that there were very often no other indications of the disease.

*Causes.*—These are ascribed to a variety of external circumstances, such as soil, pasture, geological formation, altitude, weather, and the influence of ticks.

I. *Influence of pasture, geological formation, and altitude.*—The late Mr. Brotherston, Kelso, who made an extensive examination of the geological formation of loup-ill districts, came to the conclusion that it is found on very different soils, geological formations, altitudes,—close to the sea, as in Skye, and on soils and formations so far apart as the Lower Silurian, the Upper Silurian, the Old Red Sandstone, through the Carboniferous to the Basalt of the Tertiary period, and that it existed equally



upon all these, provided old grasses were allowed to remain on the ground. (By old grasses is meant the growths of the last and previous years.)

Mr. Brotherston was the first to promulgate the opinion that the cause of the disease was contained in the withered grasses, as these grasses were extensively covered with a variety of micro-fungi; and that these fungi acted upon the animal body in a manner similar to that induced by ergot; that, in short, "louping-ill" was a species of ergotic intoxication or nervous excitement. This was a very reasonable conclusion, for, as before stated, the disease was not found unless portions of the ground were more or less thickly covered by grasses of last year's growth, all of which were covered with micro-fungi.

It was important to set this question at rest, and on 9th May 1882 four one-year-old sheep were obtained from a district in which louping-ill was unknown, and were fed at the College up to the 15th day of June, thirty-six days in all, upon these withered grasses, carefully collected by Mr. Brotherston in the Hawick district. After that, six ewes and lambs were added to the number, and fed upon the same grasses, and remained quite free from disease. Later on, the grasses were mixed with ergots, but no effect was induced beyond a slight loss of appetite and some degree of purging.

By introducing a small portion of fungus-covered grass into a cultivation fluid, long thread-like filaments could be grown, but these differed from what is to be hereafter mentioned, and the above experiment almost conclusively proved that neither the grasses nor the fungi then seen were capable of directly inducing the disease.

Doubtless animals fed upon such poor food, particularly if compelled to range over the hills in search of it, are rendered feeble and incapable of resisting the influence of disease-exciting causes, and in consequence the mortality is thus rendered much greater than where the pastures are richer and more nutritious.

II. *The influence of the weather.*—The minority of those acquainted with the disease hold the opinion that a sudden change of weather of any kind, whether from good to bad or from bad to good, is sufficient of itself to induce the disease; whilst others maintain that it appears only during the prevalence of cold east winds.

These opinions are entitled to some consideration, as it is an actual fact that the mortality is very much greater after a sudden change, particularly—and in every instance that came under observation during the investigation—a change from warm to cold and inclement weather.

But neither change of weather nor exposure to its inclemency are of themselves sufficient to induce the disease, nor does the latter in any of its phases simulate an ailment brought about by climatic influences; and the only conclusion that can be deduced from the above premises is, that inclement weather, by its debilitating influences, predisposes to disease, and causes many animals to succumb to what otherwise might have passed off without very grave consequences.

One flockmaster observed that he believed the great majority of the sheep in an infected district had the disease once in their lives in greater or less severity during the season; that if the food were good and the weather favourable, but few became seriously ill, and fewer died; but if the weather became suddenly cold or wet, the disease was able to make a deeper impression upon the animal body, and many then succumbed; and further, if the disease were due to the weather, why was it confined to certain districts, whilst in others equally cold and exposed it was entirely unknown?

Being satisfied that the grasses, geological formations, and weather were insufficient to cause the disease, one other probable source of it remained to be considered, namely—

III. *The influence of ticks (Ixodes).*—The belief is general amongst all acquainted with louping-ill that it is closely allied to ticks; the majority of farmers, indeed, believing that the parasites, by fixing themselves in the skin and sucking the blood, cause such extensive irritation, pain, and weakness, that the animals perish from these causes, and that the convulsive spasms and paralysis are merely symptoms of irritation and debility. A very brief acquaintance with the disease, however, compelled the author to conclude that, no matter how closely allied the tick and the disease were, irritation and loss of blood had little or no connection with the disease under consideration; for in many instances but few of the parasites were found on some of the diseased sheep, whilst many healthy ones were swarming with them, and seemingly feeling but little dis-

comfort. It was, however, noticed that when they were very numerous on lambs, such lambs presented the disease in an aggravated form, as if the dose of the disease-inducing material had been a strong one. The belief in the influence of the tick, or its close connection with the disease, being so general, it was necessary to consider this point very carefully, with the view of determining if any relationship existed between the parasite and the disease. The result of observations on this point was confirmatory of the popular idea; for it was found—1*st*. That where louping-ill existed there the tick was sure to be: 2*d*. That where the parasites were absent there was no louping-ill; and it is a remarkable fact that upon ground having a westerly or southern aspect ticks and louping-ill prevail, whilst on adjoining grounds with easterly or northerly aspects, upon which the grasses are short, thus providing no cover for the ticks, the disease is rare: 3*d*. That the appearance of the parasites on the sheep in April and early summer was concomitant with the annual outbreak of the disease: 4*th*. That in Skye, where it is

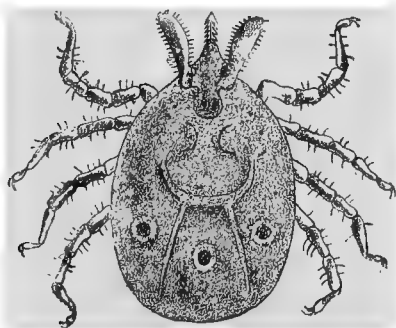


FIG. 58.—The Tick.

said the disease appears both in spring and autumn, the tick being also there in autumn as well as in spring.

Ticks are occasionally found on land free from louping-ill. From this it must be concluded that the parasites are the conveyers or inoculators of a *virus*. By improvement of the land by ploughing and liming, by artificially feeding the sheep, and destroying or otherwise disposing of old grasses—the natural

covers of the tick, as will be shown hereafter—louping-ill has been entirely eradicated.

The tick is a true blood-sucking parasite, belonging to the family *Ixodidae*, the class *Arachnidae*, and order *Acarinae*. The mouth of the tick is provided with a serrated beak or rostrum, which enables it to pierce the skin, and retain its hold very firmly, and almost without effort, as the barbs or serrations point backwards.

The species are very numerous, but the best known are the *Ixodes ricinus*, dog-tick, the *Ixodes reduvius* and *Ixodes plumbens*, sheep and cattle tick. Those found during the investigation were recognised by Mr. Moore, British Museum, as *Ixodes ricinus*, *erinaceus*, and *marginatus*.

During this investigation we found ticks upon every variety of soil, and at different altitudes—close to the sea level in Skye, and at a height of 2000 feet in the inland districts; in fact they will be found as high as sheep will go, provided always old grasses are abundant to afford cover, as they are thus protected from injury, and are left at comparative rest during the periods they are not obtaining blood from the animals which they attack. Prompted by hunger, they quit their hiding-places, and attack any animal that may come in their way, by plunging their beaks or sucking proboscides deeply into the skin, particularly about the face, root of ears, lower parts of breast, between the thighs, in fact into those parts of the skin covered more with fine hair than wool.

Thus fixed, they will hang for days together sucking the blood, until their bodies become distended to eight or ten times their original size; they then quit their hold, fall to the ground, and lay their ova, which remain dormant until next spring, and, shortly after they are hatched, they most probably attack the sheep, and after they once bite remain firmly fixed in the skin.

It was a noticeable fact that when dead or dying sheep were examined the ticks were found already dead, or in a dying condition, in almost every instance of louping-ill.

Rabbits, ferrets, stoats, weasels, and more rarely hares, are attacked by ticks, but there are no instances recorded of these animals becoming infected by louping-ill. This is probably due to these hosts proving unsuitable habitats for the development of the organism inducing "louping-ill," but horses are

said to present symptoms of the disease during the louping-ill season.

When they first leave the ground to attack animals they are very small, sometimes no larger than a pin's head, and when very numerous make the part quite black by their numbers, and they adhere so closely that scraping them off will tear up the skin.

Being satisfied that the parasites had a close connection with the disease (the proofs that they were co-existent upon certain lands, and simultaneous in their appearance, being overwhelming), but failing to see that they caused grave disease and death by inducing irritation and loss of blood to any great extent, it was necessary to discover if any organism existed in sheep dead from louping-ill, and if so, to trace its source of origin, indeed to demonstrate if such an organism was common to the tick and to animals affected with louping-ill. This was a matter of some difficulty, as in the majority of the animals examined after death no really characteristic lesions could be detected.

In many, all parts of the body presented the appearances of health, death having been evidently due to intense nervous irritation.

In some, of course, it will be understood that there were signs of other diseases, but these in reality had no connection with the animal's death, and we are now referring to louping-ill pure and simple. In some instances there is a jelly-like formation within the spinal canal, sometimes extending in a more or less uniform layer from one end of the canal to the other, but rarely extending within the cranium. It is sometimes in patches here and there, particularly embracing the roots of the spinal nerves; generally there is a large patch in the lumbar region, then smaller ones scattered along the dorsal region; often a large patch at or about the junction of the cervical and dorsal, and very frequently a large and much redder patch immediately posterior to the foramen magnum, or entrance into the brain cavity. This jelly is seen both in lambs and in aged sheep in great perfection. It was first seen in sheep by me at Langholm on 20th May 1881, in a four-year-old ewe. I have also seen it in oxen, as well as in sheep and lambs. It is, however, not constant, even in cases that

have been unwell for several days, whilst in others that have been down three or four days it is abundant. I make particular reference to this jelly, as some to whom I have shown it have asserted that it has no connection with the disease, that in fact it is a myxomatous condition of the fat surrounding the spinal cord,—a condition, they say, not inconsistent with health in young animals; and that what I considered mycelia, the development of which within the body being supposed to be an impossibility, were nothing more than capillaries, and the spores blood corpuscles.

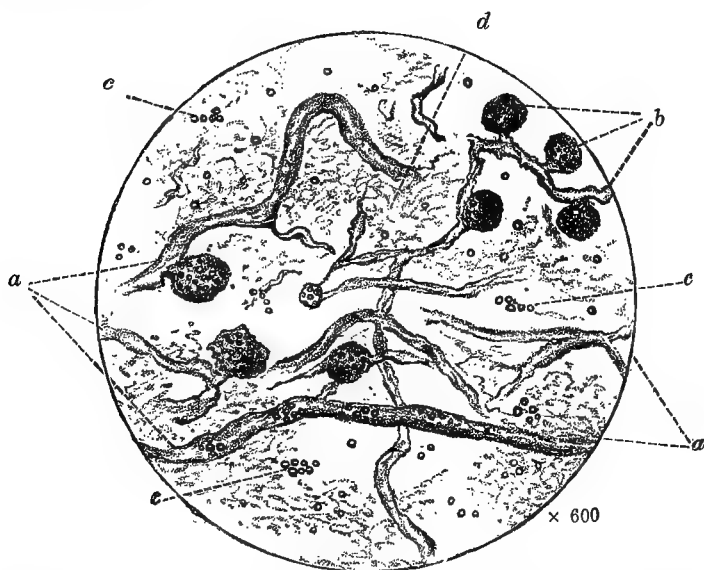


FIG. 59.—Microscopic Views of Gelatinous Matter from Spinal Canal containing the Organism in its most advanced stage. (Drawing by Dr. Jas. Hunter.)

*a.* Spore-bearing filaments.  
*c c.* Free spores.

*b.* Peculiar globular masses of spores.  
*d.* Micrococci or spores.

This jelly is a slimy tenacious substance, of a straw colour, and, as seen by the naked eye, lying external to the dura mater, and when examined microscopically is found to contain an organism, presenting the appearances seen in the illustrations.

Figures 59 and 60 have reference to the appearance seen

in fresh specimens of this jelly under powers of 600 or 700 diameters, when the most characteristic structures are those to which I have applied the term mycelia, and which, as stated above, were considered to be capillaries by some *savants* to whom I had shown the specimens. At *a* in figures 59 and 60 are seen these filaments in various forms; they appear to consist of an extremely delicate limiting membrane (appearing better defined in filaments of medium size than in the larger ones) of highly refractive semi-fluid contents, and, at intervals, masses of spores of a still higher refractive power than the fluid in which they lie.

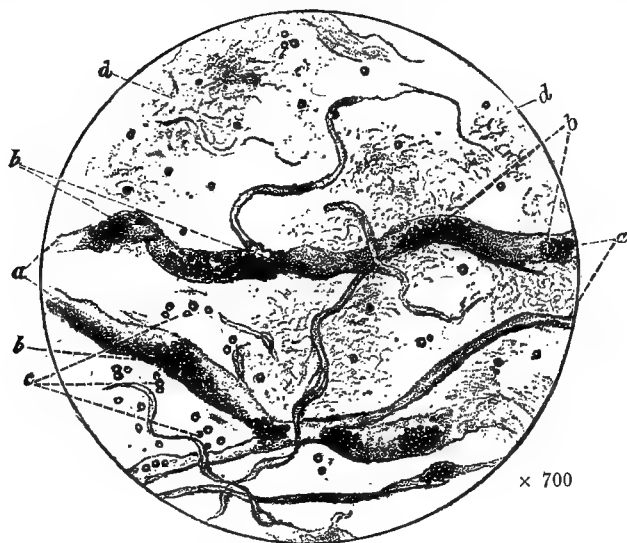


FIG. 60.—Microscopic Views of Gelatinous Matter from Spinal Canal containing the Organism in its most advanced stage. (Drawing by Dr. Jas. Hunter.)

*a a.* Filaments.

*b b.* The spore masses in the filaments.

*c c.* Free spores.

*d.* Micrococci or spores.

These spore-masses are seen at *b* in both figures, and at *c* there are a few free spores outside the filaments. Figure 60 shows the appearance I have most commonly observed, figure 59 represents a less common appearance, where they resemble berries upon short stalks.

As arguments against these mycelia or filaments being capillaries, I would point to their peculiarity of branching, the

minute size of many of these branches, their behaviour on staining; and further, that the contents, instead of being shrivelled blood-corpuscles, differ very much from such bodies in their uniformity of size, smoothness of outline, and high refractive power; but, finally, the appearance of identical filaments in fluids from cultivation of the blood and spinal fluids of sheep affected with this disease sets aside completely any possibility of their being capillaries.

The fresh spinal subarachnoid fluid obtained from spines in which the jelly or muck is absent, when dried on a glass slide and stained with methylaniline, is seen to contain a considerable number of bodies, like those marked *a* in the illustrations, and some epithelial scales, but no motile or rod-like bacteria or bacilli. When a minute drop of this clear and fresh spinal fluid is cultivated in mutton broth, even at the ordinary temperature of a room, it gives rise to the following series of changes:—first, in about eighteen hours the fluid is seen to be more or less turbid, and if examined microscopically under a good light, and with power over 600 diameters, it will be seen to be crowded with extremely minute organisms of a cylindrical or club shape, with slightly rounded ends, these cylindrical rods being either free or attached in chains. Some of the rows are single, some double, and a few are triple or even quadruple.

The single free rods are motile, and stain readily with methylaniline blue or violet, and when so stained are easily observed.

Figure 61, drawn from a specimen procured from a cultivation in mutton broth of spinal fluid from an affected sheep, shows these rods free and in several states of development, as well as masses of zoogloea with bacilli embedded therein.

A few hours later, longer chains are developed, and rod-like bodies having active motion are very abundant, whilst at other points distinct mycelia or spore-bearing tubes identical with those seen in the glutinous matter from the cord are visible, and masses of zoogloea with micrococci are seen scattered throughout the field,—figures 62 and 63, *c e*.

To gain an idea of the individual character of these rods, and also of their attachment in single or multiple chains, I refer the reader to figures 63 and 64.

A free rod, as at *d*, figures 63 and 64, presents a some-



what cylindrical outline, slightly rounded at the extremities, with a bright line (due to refraction) running down the centre, and slightly swollen out at either end. (The appearance of this line is less given by the drawing than are the other characters.) The middle part of each rod is a little constricted, but much less so than *Bacterium termo*; its length is about midway between that of *Bacterium termo* and of *Bacillum anthracis*, and the rounding of its ends is also intermediate between these two, but more nearly approaching the characters of the latter.



FIG. 61.—Cultivation from Spinal Fluid. (Drawing by Dr. Jas. Hunter )

The character of the chains or rows of rods may be seen in figs. 61 to 64, the former under 600 diameters, showing the general appearance of single and double rows, the latter of double rows under a higher power (1200) and in various stages of detachment.

These chains present very much the appearance seen in many low forms of vegetable life, only differing in point of size. Figure 61 shows that in the double rows, so long as the rods are in lateral contact, clear division lines mark the chains transversely at intervals corresponding to the lengths of two rods, and that also less distinct cross lines are visible midway between these, marking off each bundle, as it were, into four. The single

chains often present here and there an occasional filament in position alongside (see figures 63 and 64). In all of the figures are to be seen masses of zooglœa with embedded bacteria, as well as many free bacteria in the intervals. In figure 64 many of these seem to have been breaking up into *débris* (these are shown in fainter lines); indeed it was noticed that by allowing putrefaction to take place the appearance of the *Bacterium termo* was always rapidly followed by this result, and finally by the total disappearance of the bacillum under consideration. Perhaps more interesting than the appearance of the rods in all the cultivation fluids, whether from the spinal fluid or from the blood, is the appearance of these filamentous tubes marked *e* in figures 62 and 63, as proving the identity of the organisms seen in the jelly from the spinal canal, and those developed from cultivation of fluids from the affected animals, as well from the bodies of sheep ticks, as will be seen from what follows.

An examination of the blood alone gave no very satisfactory result. Here and there, however, spore-like bodies were seen: these, however, were neither numerous nor constant; but by inoculating mutton broth with a minute quantity of fresh blood a result was obtained, but not in all instances, exactly identical with that resulting from the cultivation of the spinal fluid. In the course of from twenty-four to thirty-six hours the cultivation fluid was seen to be more or less turbid, and on the microscopic examination of a stained specimen free bacilli were seen in great abundance; a few were also seen in rows. On the third or fourth day mycelia, with spores in their centre or covering their walls, became visible, as well as masses of zooglœa with numerous embedded bacilli and micrococci, as seen in figure 62.

The microscopic appearances of the jelly-like substance found in the spinal canal, and the development of the same mycelium, and spores with motile bacilli from the blood, &c., although not as yet clearing up entirely the life history of these organisms, showed that a new discovery had been made, and that the disease in question was due to the irritation of the nervous system through the spinal cord, in the fluids of which this organism seems to find a suitable nidus for complete development.

The experimental feeding of the sheep on fungoid grasses having given no result (p. 669), the author had but one resource left by which he hoped to determine whether the tick conveyed

the disease to the sheep or not, and for this purpose ticks from diseased sheep were introduced into the cultivation vessels containing the mutton broth, and the results were most conclusive. In a few days the fluid became turbid, and was found



FIG. 62.—Blood Cultivation from Diseased Sheep. (Drawing by Dr. Jas. Hunter.)

- a. Multiple rows of rods or bacterioid segments (pathogenic mould).
- b. A few rods in a single row.
- c. Mass of zooglæa with micrococci.
- d. A single rod.
- e. Mycelium with spores.

to contain numerous bacilli or rod-like bacteria, in no respect distinguishable from those got in the fluids from the diseased sheep; and further, that the peculiar filaments with spores in their interior were also seen (figure 63, e).

The strawish-coloured mould which soon covered the ticks, when stained, dried, clarified with oil of cloves, and mounted in balsam, presented the appearances seen in figures 63 and 64.

Now this proved nothing beyond the fact that the organism was in the tick; but whether the tick received it from the sheep, or the sheep from the tick, was a point yet to be determined, and for this purpose ticks were obtained from the grass and perfectly healthy sheep, and treated in the same way. The con-

clusion arrived at was that the organism was communicated by the tick from louping-ill districts to the sheep, for the same organism was found to be developed, but not quite so quickly, in the cultivation as in that containing the tick from sheep suffering from louping-ill.

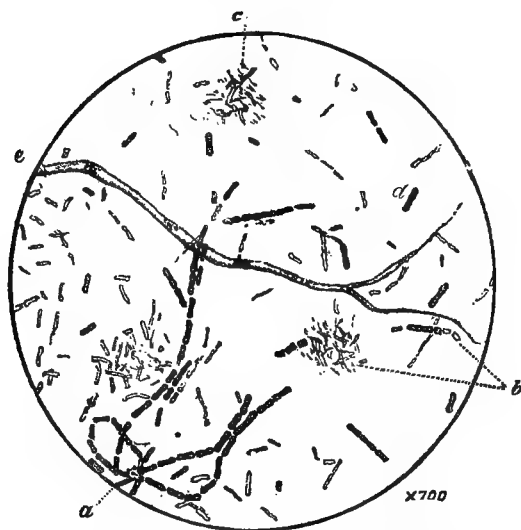


FIG. 63.—Cultivations from Blood of Tick. (Drawing by Dr. Jas. Hunter.)

- a.* Single rows of bacterioid segments with traces of a double row remaining.  
*b.* A few rods seemingly imperfect.  
*c.* Zoogloea.      *d.* A perfect and entire rod.      *e.* Mycelium.

This last experiment, repeated over and over again, proved the fact that the ticks experimented with contained the spores of the organism, and were capable of infecting sheep; and that the reason that all sheep were not infected was simply the fact that many sheep were able to resist the invasion of the organism, or suffered so slightly that they were not observed to be ill.

Many sheep even badly affected recover from louping-ill, and it is fair to conclude that the organism has but a transient effect upon many others; that in fact, as is the case with other diseases, many are able to throw it off, and are most probably by this inoculation rendered proof in many or even the majority of instances against any further infection. This conclusion is

borne out by a circumstance well known to flockmasters and shepherds, namely, that sheep reared in districts free from loup-ing-ill are much more liable to take the disease, when subjected to its cause, than those reared where the disease and ticks prevail.

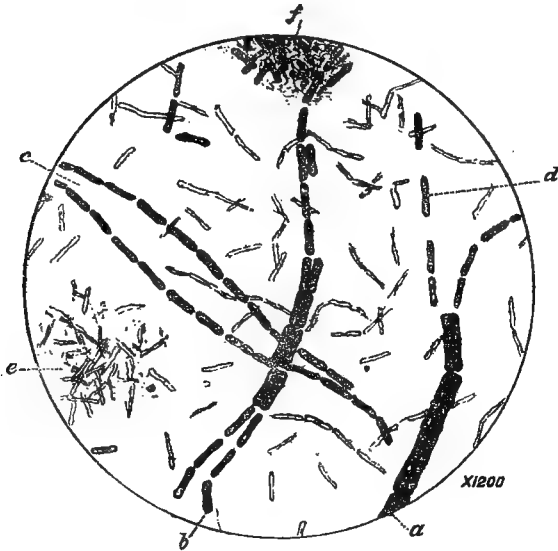


FIG. 64.—Cultivations from Blood of Tick. (Drawing by Dr. Jas. Hunter.)

- |   |  |
|---|--|
| <i>a.</i> Double row splitting into two single rows, which separate longitudinally. | <i>d.</i> A single rod.                |
| <i>b.</i> Double row in another stage of division.                                  | <i>f.</i> Zoogloea with free segments. |
| <i>c.</i> Two single rows lying nearly parallel.                                    |  |
| <i>e.</i> Zoogloea with disintegrating segments.                                    |  |

It will be noticed that the mycelia or filaments and bacilli or rods differ in size, and to some this seems antagonistic to the view that it is a specific organism.

It must, however, be remembered that these different appearances are due to the varying ages of the growth, and that the organism seems only to arrive at maturity in the spinal canal, where, in the myxa sometimes there found, it may be seen in its highest development, and it is not denied that the organism may be multiple.

Dipping the sheep with any of the recognised sheep-dips will also destroy the ticks, but this procedure cannot be very con-

veniently effected at and about the lambing season, and if lambs were dipped, great care would have to be exercised that the dip be not too strong, or else the remedy might prove worse than the disease.

The great question, however, is, can ticks be destroyed and the disease prevented? and I think I am in a position to answer in the affirmative.

It must be borne in mind that, as already stated, ticks are only found where the last and previous year's grasses are rank and afford cover for the parasites, and the remedy for the prevention of the disease is to eat down if possible, cut, burn, or otherwise remove, during the autumn all the long grasses of the previous summer's growth.

Putting aside the problem which is yet unsolved, namely, the possibility of the louping-ill organism having to pass one of its stages of existence as a micro-fungus upon some grass (such as the *Nardus stricta*, which is found in great abundance on the poor soil in such localities) before entering the body of the tick, or whether it is hereditary, as it were, in the body of some ticks, leaving the pasture bare, and subsidising it with hay or corn, recommends itself to the careful consideration of all interested in sheep, for it is a fact placed beyond all doubt, that by rendering the pastures bare the covers and hatching-places of the ticks are thus destroyed. In confirmation of this conclusion, I take the liberty of giving the experience of an Ettrick sheep-farmer, Mr. Nichol, Crosslee and Newborough on Ettrick, who was visited by Mr. Brotherston and myself on 24th June 1882.

Mr. Nichol stated that he entered the farm of Newborough twelve years ago, and that the louping-ill was then very bad. Between Whitsunday term (May 26th) and August he lost ten per cent. of his lambs—thirty-two out of sixteen score—besides several ewes. He thought the farm was not worth having. The parks were all in rough grass. Next year he bought some cattle, and far more the following year; ate, cut down, and even burned the old grasses; drained and limed the parks, using six tons of lime per acre to the light and eight tons to the heavy land. He continued to eat, cut, and burn down the grasses as bare as he could, and both the "louping-ill" and the ticks became scarcer and scarcer. The third spring after he entered the farm it was very cold and sleety up to and after the "cutting" time. Ticks were very much scarcer than they had

been before, and louping-ill was very much diminished with him, whilst on other farms it was, as might have been expected from the state of the weather, very bad. He thought, but wrongly, as he has now found out, that the wet, coarse weather was the cause of the scarcity of the ticks.

In six years both louping-ill and tick had both almost entirely disappeared—an odd one now and then—and he has had scarcely any since.

He has kept extra stock, both of cattle and sheep, during summer. The number of cattle kept has increased from forty to one hundred and twenty, which were kept during the summer of 1881. The cattle are sold about October, but about thirty head are kept on the hills during winter, part of them being tied at night. Last year Mr. Nichol stated, "You may believe the pastures were eaten to the bone." During our visit, however, we found that the grasses were more luxuriant and of much better quality than on any neighbouring land.

Mr. Nichol very quaintly remarked, "Those who can afford to let their sheep die, do so, but I cannot afford this, so give corn."

One farmer whom I know very well congratulated himself that his heavy loss had taken away all his weakly sheep, and he had now a good strong stock to breed from, stock that would stand anything, even starvation.

One moment's rational consideration of this matter would open the eyes of even the most prejudiced, and convince them that starvation never leads to strength, but to the converse condition, and that a strong healthy stock, although able to live through a hard winter without any food but what they can scrape up, are rendered weak and debilitated; and that the mortality amongst the ewes is enormous during the lambing season, that many dead lambs are brought forth, and that where both mother and offspring survive, the latter often dies from starvation, the ewe being unable to afford it its natural sustenance. It must also be borne in mind that exhaustion of soil, micro-fungi on grasses, ticks, poverty, and louping-ill are co-existent.

In conclusion, I would earnestly recommend all who may read these observations to carefully consider the suggestions laid down, to eat up or destroy the old grass, which, when left on the ground, becomes a source of mischief not only in

itself, but by affording a cover and harbour for parasites, which I think have been proved to be the source of enormous losses.

With regard to the ked, *Melophagus ovis*, found upon all sheep, and having no connection with the disease under consideration, it was discovered that those removed from sheep suffering from louping-ill contained the germs of the organism, and that it could be cultivated as readily as that of the tick itself, whereas, when it was removed from sheep healthy and not upon louping-ill land, no results were obtained, although several experiments were made. The fact that ticks induce more diseases than louping-ill is now fully recognised (see Texas Fever).

### FURTHER INVESTIGATIONS, 1894-6.

#### *Louping-ill Inoculations at the College.*

1894,

- May 2. Sheep inoculated at 3.25 P.M. with cultivation from sheep killed at Brown's farm, Dalmally, whilst suffering from louping-ill. Temperature 103° F.
- „ 3. Temperature 102° F.
- „ 4. Partial paralysis of the fore limbs; convulsive twitchings or “trembling”; foaming at the mouth. Temperature 102° F.
- „ 5. Complete paralysis of the fore limbs; partial paralysis of the hind limbs; convulsive movements, &c. increased. Temperature 104° F.
- „ 6. Sheep died, 8 A.M. *Post-mortem* revealed louping-ill lesions.

On the same day, May 2, from ten to fifteen living ticks were placed on each of the remaining sheep, six in number. These were examined on the following morning, when it was found that the ticks had almost entirely disappeared; they were re-examined daily until June 2, and, giving no evidence of infection, all the ticks having evidently dropped off without becoming adherent to the skin of the sheep, the following experiments were made:—

#### *Inoculations with Cultivations from Ticks.*

- June 2. Sheep No. 1 inoculated, 2.30 P.M. Temperature 104½° F.
- „ 3. Temperature 103° F.
- „ 4. Temperature 104° F.
- „ 5. Temperature 104° F.
- „ 6. Temperature 104° F. Results negative.
- „ 9. Sheep No. 2 inoculated, 2 P.M. Temperature 104½° F.
- „ 10. Temperature 105° F.



- June 11. Temperature 105° F.  
 „ 12. Temperature 103° F. Results negative.  
 „ 16. Sheep No. 3 inoculated, 1 P.M. Temperature 103° F.  
 „ 17. Temperature 103° F.  
 „ 18. Temperature 102·4° F.  
 „ 19. Temperature 102·6° F.  
 „ 20. Temperature 102° F. Results negative.  
 „ 16. Sheep No. 4 inoculated with tick cultivation in egg albumen, 1 P.M.  
 Temperature 103·4° F.  
 „ 17. Temperature 103° F.  
 „ 18. Temperature 103° F.  
 „ 19. Temperature 103·2° F.  
 „ 20. Temperature 104° F. Results negative.  
 „ 20. Tup No. 5 inoculated, 4 30 P.M. Temperature 103° F.  
 „ 21. Temperature 105° F.  
 „ 22. Temperature 103° F.  
 „ 23. Temperature 103° F.  
 „ 24. Temperature 103·6° F.  
 „ 25. Temperature 103° F. Results negative.  
 „ 29. Sheep No. 6 inoculated *in vein*. Temperature 104° F.  
 „ 30. Temperature 104° F.  
 July 1. Temperature 103·8° F. Results negative.  
 June 29. Sheep No. 1 reinoculated *in back* (subcutaneously). Temperature  
 103° F.  
 „ 30. Temperature 103·8° F.  
 July 1. Temperature 103° F. Results negative.

The attempt to induce the disease by placing the ticks upon healthy sheep, and by inoculations with the organism cultivated from ticks, having proved unsatisfactory, and the season being too far advanced for further experiments, I determined to adopt another line of procedure during the season 1895. I may here state that all the experiments with living ticks had hitherto given negative results: ticks, except small ones and those which had not yet become adherent to the sheep, could not be induced to cling to a fresh host; no matter how many fairly or fully grown ticks were placed on a sheep, they would disappear in a few hours.

I then determined to send sheep during the following year to tick-infested districts; to muzzle these sheep in order to prevent them from grazing; to feed and water them indoors with food sent from the College; and to allow them to remain in the infested district until infested with the ticks, then have them returned to the College for examination and further study.

“*April 21, 1896.*—We visited Mr. Hamilton, Leithen Hall, and an adjoining farm, occupied and owned by Mr. Carruthers.

He removed there from the south of Scotland (the Hawick district) about fifty years ago, and placed a large number of sheep from that part on his new farm. The mortality was very great, quite 30 per cent. He reduced this percentage by limiting the introduction of fresh sheep, and devoting his energies towards the increase and retention of sheep home bred. The loss was thus reduced to 10 per cent; and when he became the owner of the land he limed and drained to such an extent that at this date a case of louping-ill is very rare. He is a firm believer in 'no ticks, no louping-ill.' He frequently witnessed in the old days bullocks affected with the disease. He still washes his sheep with Bigges' Dip in solution, and lays down the rule to be good to the land, and louping-ill will disappear wherever it has been seen.

"At four A.M. on the morning of the 22d April we accompanied the shepherd to the hill, and found some 600 ewes and their lambs all in good health. About thirty six hours previously the shepherd had found a hogg dead that for two days previously showed very pronounced symptoms of louping-ill. He had, after skinning it, preserved the carcass for our examination in a cavern on the hill. We found it to be cool and quite fresh, with the head cut off. This we regretted, but he stated that a large quantity of fluid had escaped from the cranial cavity on section. At nine A.M. we (having had the carcass and head carried to the farm-house) made an examination of all the internal organs, also of the brain and spinal cord. The jelly-like exudate was present in great abundance, particularly in the lumbar portion of the spinal canal; the spinal cord was inflamed in patches along its whole length, but the jelly-like exudate was confined to the lumbar region.

"The test sheep having arrived—they having been driven from the station with a mouth-piece on each, which effectually prevented them from feeding—the muzzles being removed, they were fed with oats and hay and watered, also rested for a few hours. The mouth-pieces were then re-applied, and about one o'clock they were turned on to a field where ticks were known to exist. About ten A.M. the following day they were returned to the outhouse to be fed and watered. A few minute ticks were seen about the inner parts of the fore legs. At one P.M. they were again turned out with their mouths securely covered. Every day until Tuesday, the 28th, when the sheep were re-

turned to the College, this system was carried out. One of the ticked sheep died on 10th May and another on 12th."<sup>1</sup>

On May 13 ten sheep, out of eleven obtained from Mr. Skirving, were sent from Edinburgh to Leithen Hall, as I supposed the ticks would now be more numerous. They safely arrived, were muzzled in the usual way, and remained there until the 21st, when they returned apparently healthy.

The student, Mr. Morgan Williams, who accompanied these sheep, wrote to me as follows:—"The sheep were all muzzled, and examined for seven days, but owing to the early season this year very few ticks were found on the farms, neither were there any sheep affected with louping-ill during the above period. Out of the ten sheep two only were found with ticks on, and, as far as could be seen, there were only three ticks on each. Nothing abnormal was detected on these from the time they came under my charge until they were brought back to the College. Mr. Hamilton informed me that fewer of their sheep were infested this season with ticks, and consequently fewer were affected with louping-ill; that they were all dipped this season, as recommended by Professor Williams. He also informed me that a top-dressing of salt, 10 cwt. to the acre, gratuitously supplied by Mr. Thompson, V.S., Aspatria, was applied to one park, which had been previously badly infested with ticks; but since the application of the salt the sheep cannot be kept away from the dressed park. But Mr. Hamilton could not inform me whether this dressing had made any difference as regards the ticks, because, as above stated, the sheep were dipped, and they did not find any on the sheep after the application (dip), irrespective of the part of the farm the sheep were grazed on."

On their return to the College, 21st May, these ten sheep were carefully examined; but no ticks could be found upon any of them. They were carefully watched, and their temperatures taken daily until 27th May, when four, including the one retained at the College, were inoculated with cultivation material from the spinal canal of the ticked sheep which had died on the 12th. The cultivation material was filled with the well-developed organism. One of these sheep died on May 29, having presented very acute symptoms, which we supposed were due to some septic blood-poisoning; but on making a *post-mortem* examination, the condition of the spinal cord and

<sup>1</sup> Report by Mr. R. Moir, M.R.C.V.S., who kindly took charge of the sheep.

brain left no doubt in our minds as to the true nature of the disease. Although there was no jelly-like exudate in the spinal canal, the cord itself was inflamed in patches, and the organism could be detected microscopically and cultivated in the usual way. On 1st June a second inoculated sheep died, and on 5th June another died. Thus three out of four inoculated on 27th May died. The fourth remained healthy.

On May 5 I visited Mr. James R. W. Wallace's farm of Auchenbrack, near Thornhill, and there met Professor Wallace, who takes a keen interest in this question. We found several sheep and lambs affected—some dying, some recovering, and some dead; but Mr. Wallace thought the mortality had not been as great as usual. The symptoms during life and the *post-mortem* conditions revealed were undoubtedly those of louping-ill. No experiments beyond tube cultivations were made, but it was arranged—Mr. Wallace generously offering to perform the experiment free of expense—to buy about a score of sheep and place them muzzled on that part of the farm where trembling and ticks committed the greatest havoc. The sheep, nineteen blackfaced hogs, were bought on May 13, and fourteen of them were muzzled and turned out during the day, and described by Mr. Wallace as follows: "13th May.—Will turn out fourteen of the ewe hogs on the hill on which, there is trembling, with the muzzles on, and take them in again in the morning, and driving them to the fields free from trembling to feed during the day. 26th May.—The sheep have been a week on, and I only see one tick each on three of them. The season being so early, I think the time is now past, as fewer of my own sheep have been affected this year; but I will go on with the hogs this week, and then turn them on sound land free of trembling for a while, till we see if anything turns up." On 1st June one of the ewe hogs died, and was sent to the College, where a *post-mortem* examination was made, but no trace of louping-ill could be detected. On 6th July Mr. Wallace wrote again as follows: "The blackfaced ewe hogs I had muzzled and put to the hill to test for trembling have all done well since they were turned out. It was rather late for this early season, so a better result might be got another year. There were not so many ticks and less trembling with me this season than usual, but some places were worse. I did not think the hogg that died, died of trembling, but I thought it better to send you the carcase, as it was one of the lot."

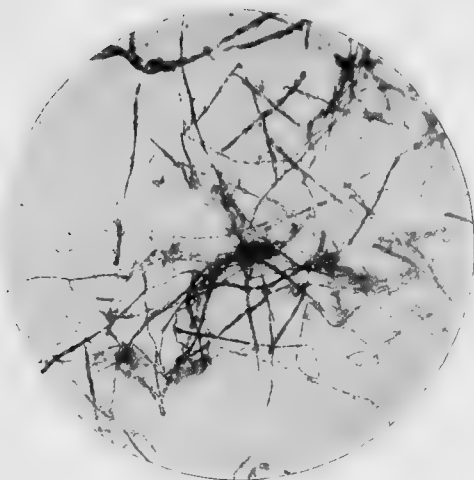


FIG. 65.—Filamentous stage of the organism in spinal canal of sheep dead from louping-ill, May, 1896.  $\times 500$ .

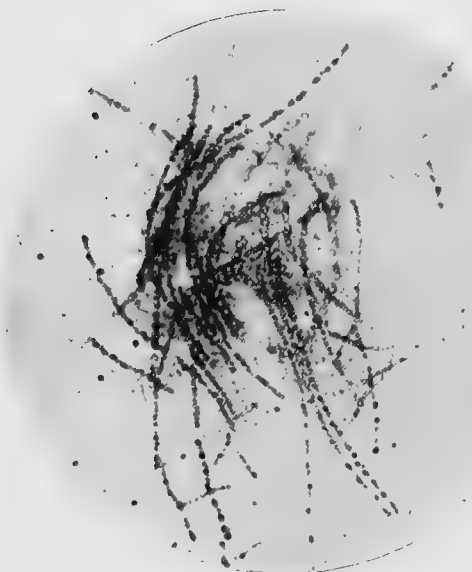


FIG. 66.—Filaments breaking up into spores. Cultivation from blood of sheep inoculated with material shown in Fig. 65.  $\times 1500$ .

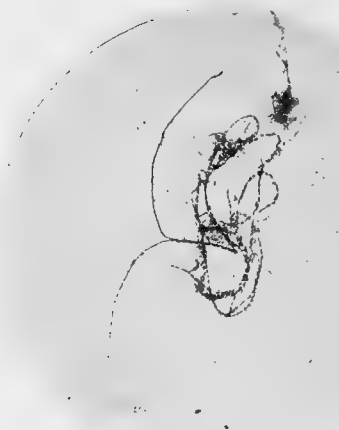


FIG. 67.—Cultivation from tick, tenth day.  $\times 500$ .

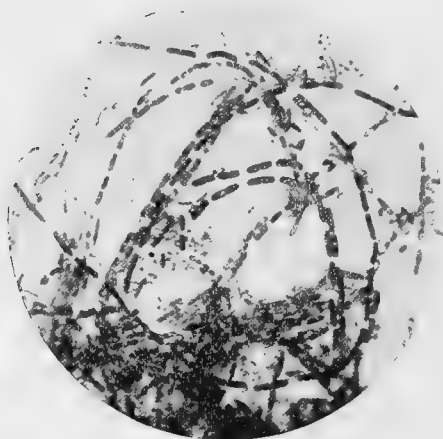


FIG. 68.—Portion of Fig. 67 under a higher power.  $\times 1500$ .

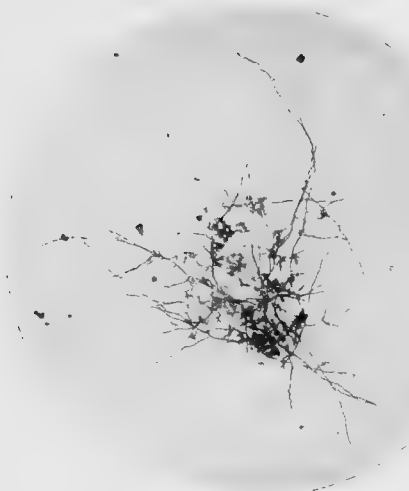


FIG. 69.—Jelly from spinal canal of Twiglees lamb, 1882.  $\times 500$ .

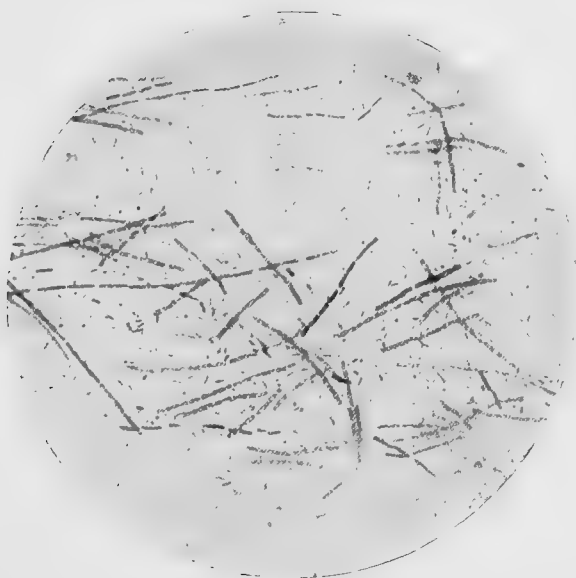


FIG. 70.—Cultivation from cardiac blood of sheep which died after being inoculated, June 5, 1896.  $\times 1500$ .

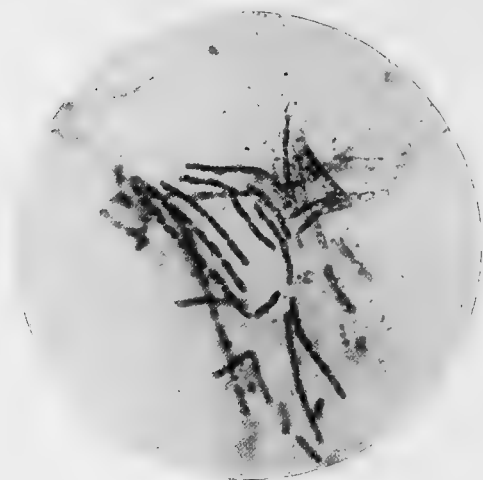


FIG. 71.—Cultivation from spinal canal of ticked sheep, which died at College,  
May 10, 1896.  $\times 1500$ .

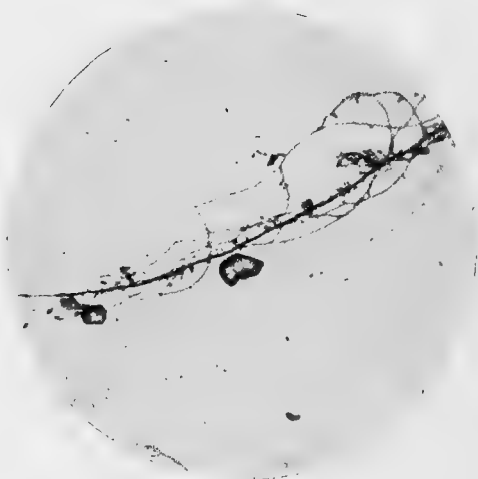


FIG. 72.—Cultivation of spinal fluid from sheep, dead from louping-ill,  
1896.  $\times 500$ .



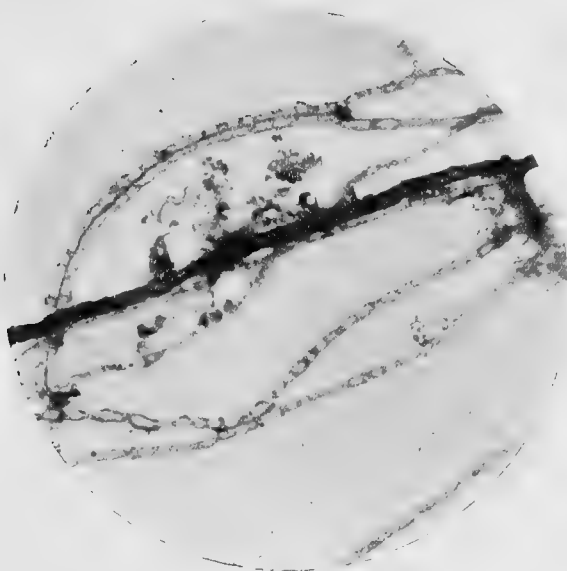


FIG. 73.—Portion of Fig. 74 under a higher power.  $\times 1500$ .

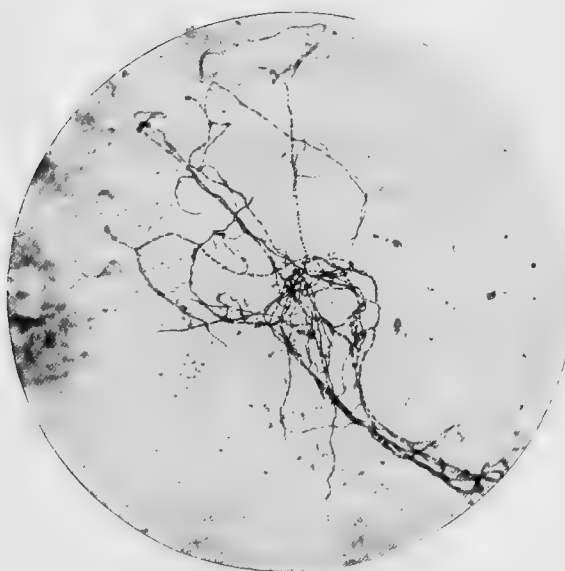


FIG. 74.—Cultivation from cardiac blood, Auchenbrack lamb (agar-agar ten days), May, 1896.  $\times 500$ .

By comparing the photo-micrographs with the microscopic drawings, both by Dr. Hunter, published in my article on Louping-ill in 1882,<sup>1</sup> it will be observed that the similarity, if not identity, is maintained throughout. From the long and exhaustive investigation (in which I have been assisted by my son, W. O. Williams, and Dr. James Hunter, and to whom my thanks are due) of this disease, I am now more convinced than ever that the organism or organisms are most probably developed in one stage as minute moulds on old grasses—in this condition, innocuous to the higher animal, until it has passed through the tick; that in the body of the tick it attains properties which are virulent when subsequently communicated to the sheep by the tick.

It will be observed that in the louping-ill organism there are met with, during its complete life history, those involving series of transitions assumed by G. Zopf and other bacteriologists to be more or less presented during the development of many micro-organisms. Leaving it, as most have done, to be decided whether or not the forms seen in such cases as the present are the exact counterparts of saprophytes, typical micrococci, bacteria, or bacilli, there is no doubt that such variations are seen during the growth of the louping-ill organism. But further, when it has been found that the inoculation of healthy animals by certain of these forms has led to the appearance of the others in great constancy under varied conditions, the identity of a series of forms can no longer be disputed. It must, however, be confessed that as yet the true relationship and significance of the individual forms have not been so fully made out as could be wished from a purely biological point of view; but the pathogenic action peculiar to the organism has, by all the usual modes of research, been placed beyond cavil. This is not to be wondered at, when it is remembered that several well-known micro-organisms have stages in their development that are by no means fully understood. Apparently the simple unsegmented saprophytic filaments are generally met with in the spinal myxœdema of the affected animal, while the higher forms of spore filaments, fully segmented and free rods, closely resembling the typical bacillus, were seen, so far as the present observations go, mostly in culti-

<sup>1</sup> See Transactions of the Highland and Agricultural Society.

vations outside the animal body. They, however, presented themselves with great constancy in cultivations of the spinal fluid and myxœdema—jelly-like material—under every possible variation, produced by change of media, place, and manner of experimenting. It must therefore be surmised that they are either various stages in the life history of the same organism, or that, as in some other diseases, multiple organisms are present. As an organism certainly more nearly allied to the moulds than to any pathogenic type previously described, it is interesting as being, so far as I am aware, the first known instance of what appears to be a mould, in one stage at least, proving capable of producing pathogenic effects upon the higher animals, and it is also interesting to note that this pathogenic property is probably acquired after passage through the body of the tick, a state of matters now found to exist in other diseases.

When this disease was first investigated by me, the influence of the tick in inducing the so-called Texas fever, and other similar sources of inoculation, had not been investigated; but now it is proved beyond all doubt that the tick disease, as well as other diseases communicated by insects, is now known in various parts of the world.

One remarkable circumstance might, in conclusion, be added, viz., that whilst in this country the tick is but seldom injurious to other animals than sheep, in other parts of the world its pathogenic effects are mostly confined to horned cattle, although it attacks human beings, sheep, horses, mules, &c.

In 1901 the President of the Board of Agriculture appointed a Departmental Committee to report as to the ætiology, pathology, and morbid anatomy of the diseases of sheep, known as louping-ill and braxy.

Professor Hamilton, of Aberdeen University (who had gained some knowledge of these diseases when on the Committee or Commission appointed by the Highland and Agricultural Society, the chief of which was Principal Williams of the New Veterinary College), was appointed chairman, and in the findings of the Committee of 1901, published in 1906, Professor Hamilton takes the sole responsibility for the conduct of the research and for the bacteriological part of it, and herewith are appended certain portions of these reports.

There seems to have been hopeless confusion to commence with, and the editor feels quite unable to accept the conclusions arrived at.

## SECTION I.

LOUPING-ILL (CHOREA PARALYTICA OVIS).<sup>1</sup>

*Recognition of the Disease.*—The symptoms at first appeared to be somewhat anomalous, and this rendered their study all the more perplexing, for while some of the animals exhibited distinct nervous spasms of the limbs and neck, or it might be almost complete rigidity, followed by more or less complete paralysis of motion, others again seemed to be in a dazed condition throughout, and speedily collapsed.

\*            \*            \*            \*            \*

We were confronted with the problem, therefore, of whether we had to deal with a single disease or whether this train of phenomena roughly comprised under the term "louping-ill" was of a two-fold nature. Had we to deal with two diseases running side by side, say, "louping-ill" proper and so-called "black-quarter"? or were the symptoms due to the same agent modified in some peculiar manner?

\*            \*            \*            \*            \*

It must be remembered and emphasised, that the sheep is subject to many other diseases, especially in the spring months, which may readily be mistaken for the disease in question, such as puerperal septicæmia or pyæmia, malignant cedema, sturdy, a form of anæmia known as "blood-rot" sometimes very prevalent in the early spring months, and, in lambs, spinal abscess with meningitis followed or not by pyæmia.

\*            \*            \*            \*            \*

It has been the practice of others before us, as it was with ourselves during this season of 1902, to slaughter most of the animals under observation when the disease was in the acute stage of its course. The reason for this is self-evident, for while the disease is at its height it might be supposed that there would be afforded the very best opportunity of detecting any lesions produced by it, and of securing the liquids and tissues in a perfectly fresh condition. As a matter of fact, however, if this method be practised, any clue to the cause of this disease will be almost certainly lost. Everything probably will be found in what seems to be an almost perfect state of health, without the slightest indication of where to look for the hidden cause of the disease. We learned by experience to renounce this method of procedure, to allow the animal to die a natural death, and to make the examination immediately afterwards. It was by so doing that the first indication of the pathology of the disease was obtained.

\*            \*            \*            \*            \*

When we slaughtered the sheep at the height of the disease, the peritoneal liquid had invariably the clear limpid character of that in health, whereas, did the animal perish in the natural course of the disease,

<sup>1</sup> So named by Williams in 1881.

it very often was turbid; but sometimes, even under these circumstances, it was quite unaltered, or appeared to be so.

\* \* \* \* \*

Microscopic examination of the clear liquid usually failed to discover anything abnormal within it. A few lymph-corpuscles such as may be met with in health seemed to be the only particular matter which it contained. The most persistent examination of it, stained or unstained, failed to reveal the presence of any micro-parasite. The turbid variety of the liquid however, was always swarming with a bacillus. This bacillus was of great size (*see* Part II., p. 28), and in a large number of instances had developed a spore, also of large size, in its interior, while liberated spores were abundant in the surroundings. Where this turbid liquid existed, the abdominal walls and, indeed, the carcase generally underwent rapid putrefaction. The abdomen became distended with gas, and the odour, which was that of putrefaction, was very much in evidence. Those carcasses, on the contrary, which possessed clear peritoneal liquid did not putrefy so rapidly, and the odour was less striking.

\* \* \* \* \*

*End of First Season at Kielder.*—The season being over, we left the Kielder district this summer (1902), after nearly two months' sojourn, with certainly a knowledge of the symptoms and morbid anatomy of the disease, of the negative character of the blood as a medium of contagion, of the difficulty of reproducing louping-ill by inoculation, and under the impression that we had to deal with two diseases, the one louping-ill proper the other so-called "black-quarter."

\* \* \* \* \*

Then, further, we had administered the contents of the stomach and intestine to normal sheep and had failed to confer the disease upon them; we had injected the same into the rectum but without positive result. All this experience had a misleading tendency, and served to divert our attention from the alimentary canal as the source of entrance of the organism.

\* \* \* \* \*

We were also imbued with the idea that the tick might be the means of introduction of a poison—a virus—but what virus no one knew. This theory had also to be run to death, its truth or falsity inquired into.

\* \* \* \* \*

*Cause of the Seasonal Character of the Disease.*—That the blood of the sheep not only possesses such a solvent action upon the louping-ill bacillus, but that this is much more in evidence during certain months of the year, and in certain individuals, is demonstrated by our series of experiments detailed in Part II., Section XXVI., p. 200. The time at which it is most bactericidal is from midsummer on to early autumn, such being the time at which the disease is quiescent. Supposing the bacillus to be in the intestine at this time, it presumably never gets a chance of passing through its walls. In the susceptible months, on the contrary,

the blood becomes an excellent medium for the culture of the organism even *in vitro*, and the bacillus will increase upon it and develop spores better than on any other nutritive basis.

\*            \*            \*            \*            \*            \*

This is evidently the key to understanding why the disease breaks out at one particular season—namely, in the spring. The blood of the sheep at that time possesses less bactericidal or protective influence than at any other.

\*            \*            \*            \*            \*            \*

But not only is this exemplified in the case of louping-ill, it holds good, even in a more remarkable degree, of braxy. During the months in which braxy prevails the sheep's blood will, as a rule, be found to be an excellent medium upon which to grow the braxy organism. It will multiply and spore upon it with the greatest aptitude, while at other seasons—spring to late summer—it is completely bactericidal. We have failed totally to grow it on sheep's blood during the summer months. Even where the admixture of bacillus with blood is made so strong that every field of the microscope swarms with the organism, not a single bacillus will be found after twenty-four hours' incubation. This, in our experience, occurs with perfect constancy, every sample of blood we have employed having given the same result during the summer months.

\*            \*            \*            \*            \*            \*

The sheep would seem, in this regard, to be a peculiarly constituted animal; it suffers from seasonal or periodic germ diseases, and the periodicity in their incidence is to be accounted for by the loss of the protective action of its blood at these particular times. This loss of bactericidal power varies with each organism, for while louping-ill and struck reach their zenith during the spring months, braxy is a disease of the late autumn and early winter.

\*            \*            \*            \*            \*            \*

### *Conclusions.*

Braxy and louping-ill form two of a group of specific bacterial diseases.

There is some amount of similarity in the symptoms of certain of the diseases of this group.

This similarity frequently results in errors of identification.

The primary habitat of the bacteria which are their cause is, in the whole of them, the alimentary canal.

At certain seasons of the year the blood of the sheep is unable to resist the invasion of these bacteria, and death ensues.

At other seasons the blood of the sheep destroys these bacteria, and at such times the animal is proof against them.

The germs of this group of diseases are picked up by the animal when feeding.

The fatal effect of these germs in the case of louping-ill and braxy may

be prevented by drenching with a culture of the respective bacilli during the period of resistance.

If the drenching be done at a wrong time of year, viz., during a period of susceptibility, death may follow as a result in a certain number.

It has yet to be shown whether drenching methods will succeed with the other diseases of the group, but the evidence, so far as it goes, tends to show that not only is this the case, but that immunity may probably be secured against two or more of them at the same time.

#### NEURITIS—NEURALGIA.

*Inflammation of the Nerves—Neuritis.*—When one considers the very delicate organisation of nervous tissue, and the extreme sensibility of the sensory nerves, a most striking fact presents itself,—namely, the rarity or almost total absence of inflammation of the nerves. The nervous substance contained within the cranium, as well as that constituting the spinal cord, is subject to inflammatory changes; but it may truly be said that, except as a result of blood alteration—as in azoturia, where the sciatic nerves, and traumatic tenanus, where the nerves connected with the wound and their neurilemmæ are often found inflamed—neuritis is almost unknown, or, if it does exist, has not as yet been correctly diagnosed.

*Neuralgia*, or pain in the course of a nerve, and recurring in some few cases at stated intervals, whilst in others the recurrence is very uncertain, is also a disease which is rarely or never met with in the lower animals. I certainly once heard of a case which presented some signs of having neuralgia of the face, but whether it was that, or pruritus, the practitioner, under whose care the horse was placed, could not tell. The symptoms were persistent rubbing of the face against any solid object for a certain period each day, with shaking of the head and great restlessness.

The principles of treatment applicable to neuralgic diseases will entirely depend upon the cause. If a nerve leading from a wound be inflamed, then fomentations and emollients must be applied; whilst belladonna should be rubbed along the course of the nerve, or, if the pain be excessive, morphia may be injected subcutaneously into the tissues of the painful part.

Should it be considered that neuralgic pains arise from a mal- or debilitated condition of the animal generally, then measures must be resorted to which are calculated to restore the body to

its normal condition, and in many cases aperients, followed by nervine tonics, such as arsenic, nux vomica, iron, or, where it is deemed necessary to soothe pain, belladonna, stramonium, yellow jessamine, or the bromides, may be administered for a short period. If pain on the course of a nerve threaten to become chronic, a smart blister will be imperatively called for.



## CHAPTER LVII.

### SPORADIC DISEASES—*continued*.

#### LOCAL DISEASES—*continued*.

##### (I.) DISEASES OF THE RESPIRATORY ORGANS.

BEFORE entering upon the consideration of the particular diseases of the respiratory organs, it will be necessary to point out certain modes of physical examination by which the diagnosis of these diseases is accurately traced.

1. *Auscultation*.—In its technical sense this term indicates the act of listening to the sounds of the interior by means of the ear applied to the surface of the body.

Auscultation may be practised *directly* by applying the ear to the part; or *indirectly* through the medium of instruments called stethoscopes or phonendoscopes. Generally *direct* or *immediate* answers every purpose; and in veterinary practice, at least, *indirect* or *mediate* auscultation is seldom practised. Auscultation, discovered by Laennec, was introduced into veterinary practice by Delafond and Leblanc.

Many of the sounds characteristic of abnormal conditions in the human being are not heard in the lower animals; hence auscultation, as well as percussion, is less satisfactory in the hands of veterinary surgeons than in those of the physician. There are various reasons for this, and not the least of them is the fact that the thoracic walls of man are much more thinly covered with muscular tissue than those of the lower animals. Again, a large portion of the thoracic cavity in our patients is covered by the scapulæ, which form insuperable barriers to examination. These, in addition to the movements of the *panniculus carnosus*, the horizontal position of the body, oblique arrangement of the diaphragm, the pressure of the digestive

organs, and very often the restlessness of the patient, render this method of examination much less satisfactory than it otherwise would be; but, notwithstanding all these drawbacks, auscultation is of immense advantage, and by it we are enabled to arrive at conclusions that would otherwise be impossible.

To become familiar with the knowledge to be acquired by auscultation much time and labour must be devoted to it, first on healthy, and afterwards on unhealthy animals; in fact the ear must be trained by long experience.

The phonendoscope can be bought at any instrument maker's, and the simpler it is the better. In applying it to the chest, its flat-shaped portion is to be accurately and firmly applied to the surface, and the opposite end in perfect apposition to the ear.

When immediate auscultation is practised, care is to be taken that the ear be accurately applied to the skin, or if the state of the surface does not admit the ear, a single fold of a thin cloth only should be allowed to intervene, as the rubbing of two folds or surfaces may create a sound and puzzle the examiner.

2. *Percussion*.—This process consists in striking upon the surface with the view of eliciting sounds, by the nature of which an opinion may be formed of the conditions of the parts beneath. Like auscultation, percussion is either *immediate* or *mediate*. The former was employed by Avenbrugger, and the latter invented by M. Piorry, who gave the name of “pleximeter” to the intervening body.

In immediate percussion, the ends of the fingers are brought together and supported by the thumb, and the parts are struck perpendicularly to the surface, or the parts may be rapped with the knuckles of the closed hand, the force of the blows being regulated by the depths of the parts to be examined, and the size and delicacy of the animal. In the cat or dog gentle blows with the tip of the middle finger are generally sufficient.

In mediate percussion, the pleximeter is generally a flat, oval, or circular piece of ivory or gutta percha, or the left index finger of the operator, which is certainly the most convenient and best intervening body which can be had.

M. Poirson of Paris recommends that percussion should be performed by means of a common sewing-thimble placed on the middle or fore finger, so as to include a small portion of air between the end of the finger and that of the thimble. The intensity of sound elicited is said to be thus greatly increased.

Some operators substitute a small hammer for the fingers, the head being made of wood, ivory, or metal, with its percussing surface covered by a softish and somewhat elastic substance, as felt, caoutchouc or gutta-percha. The more satisfactory method, and the best and simplest, is immediate percussion by the tips of the fingers or knuckles; the part percussed to be unclothed, and struck perpendicularly to its surface, otherwise the character of the sound will sometimes be confusing.

3. *Palpation*.—Touch is useful in the diagnosis not only of chest diseases but of many other ailments. By this method we detect tenderness, heat, coldness, fluctuations, tumours, and a variety of conditions indicative of disease.

If an animal be affected with pleurisy, firm pressure in the intercostal spaces will cause wincing, grunting, or even groaning.

4. *Mensuration* is recommended by the French veterinarians. It has, however, found little or no favour in this country, as it is considered that an amount of disease sufficient to alter the relative size of the sides of the chest is otherwise determined than by measurement. In the cat, dog, and cow, the intercostal spaces are expanded and dilated in hydrothorax of one side, if of long standing; and in some cases of chronic pneumonia in cattle I have confirmed the observations of Delafond, that one side of the chest is sometimes palpably larger than the opposite side.

Mensuration as applied to the chest, says Mr. Gamgee, “consists in the application of a cord or tape to the similar parts of the two sides of the chest, in order to ascertain whether any difference exists in their relative prominence. For the larger animals a tape of three or four feet long, and for the smaller, one of a foot and a half to two feet, may be employed. One end is placed immediately behind the withers, and the line carried downward to the middle of the sternum, which part is marked by a knot; a second measurement is made from the withers to the eighth rib, or the commencement of the cartilages of the false ribs, and similarly marked; a third measurement is made from the lower end of the third rib to the commencement of the cartilages of the false ribs; lastly, the tape may be carried from the posterior border of the shoulder along the middle region of the chest as far as the last rib. The same measurement may then be made on the opposite side of the thorax, care being taken that they be always made at the same stage of the respiratory act, as otherwise false

results will be obtained. It is well, indeed, to measure the different parts after both inspiration and expiration, that any difference in the expansion of the two sides of the thorax may be ascertained."

5. *Inspection*.—Some symptoms are observed by inspection only—the condition of the visible membranes, the attitudes and movements of the body, the expression of the countenance, the character of the respirations, the degree of fulness, or the converse, of the several parts. In human medicine the state of the larynx can be thus detected by an instrument called a laryngoscope, but owing to the length of the oral cavity, the dimensions of the *velum pendulum palati*, and the position of the larynx itself, the laryngoscope has not yet been made available for veterinary purposes.

6. "*Succussion* consists in grasping the thorax between both hands, and shaking it quickly so as to elicit sound. It is only useful when gaseous and liquid matters coexist in the pleura; in such cases, however, a splashing or churning sound may be heard. It will strike every one that this measure is only applicable to the smallest class of animals."—(GAMGEE'S *Domestic Animals*, page 537.) I have never practised this method of examination, nor do I recommend it, and here describe it in Mr. Gamgee's own words.

#### RESPIRATORY SOUNDS.

The respiratory sounds, normal and abnormal, detectable by auscultation, are divided into four classes, namely, Nasal, Laryngeal, Tracheal, and Thoracic.

#### NASAL SOUNDS.

In the normal condition a soft, to-and-fro, blowing noise, of equal intensity on both sides, is heard when the ear is approached to the nostrils. This sound is increased with exercise, and then bears a resemblance to the sound of a large bellows. There is no sound detectable through the parietes of the nose nor the sinuses of the head except after exercise, when a slight snore may be detected through the first and a murmur in the latter.

In some horses the nasal sound is abnormally increased with

exercise, and can be heard at a considerable distance from the animal, causing the horse to be termed a "high blower." This sound is generally much greater when the animal is gently trotted or cantered than in the gallop, and is louder in the expiratory than in the inspiratory act. The increase of sound arises from flapping or crackling of the nostrils, and has no connection with any disease. "It arises," says General Sir Frederick Fitzwygram, "from powerful muscular development in the part. If a horse so formed be pushed to its speed and continued for some time, it will be seen that it can intermit the noise at its will; and when it really becomes distressed at its pace it will have something else to do than flap about its nostrils, and the sound will then cease altogether."—(*Horses and Stables*.) I have repeatedly observed that some horses will emit this sound when they are first put into training after resting for a while, but when they are brought into condition the sound entirely ceases, and I have thought that the sound was due to flaccidity of the *dilatores nares* muscles. In other horses, again, high blowing is due to a natural narrowness of the nostrils and nasal passages. It never seems to interfere with the horse's usefulness, and, consequently, is not to be considered an unsoundness.

Snoring is diagnostic of polypus, thickening of the Schneiderian membrane, or some other obstruction to the passage of air through the nasal chambers.

A snuffling sound indicates some disease, accompanied by discharge from the nose, as purpura, glanders, catarrh, &c.

Whistling may be due to a tumefied condition of the Schneiderian membrane. This is, however, rare, and may be mistaken for a sound arising in the larynx or even in the lungs, which sometimes so retains its force within the nasal chambers as to lead one to think it originates there. Auscultation of the several parts will, however, enable the practitioner to determine where the sound originates.

Sneezing or snorting indicates an irritable condition of the Schneiderian membrane, and is present in many cases in the early stages of catarrh. Sometimes I have seen it very persistent after the subsidence of the catarrhal symptoms, or existing independently of any apparent disease, coming on in paroxysms when the animal has been at work. One fit of sneezing has

followed another, causing the animal a deal of distress. Steaming the head has succeeded in allaying it in some instances, whilst in others it has been necessary to apply a blister to the face.

*Epistaxis*, or bleeding from the nose, occurs as a symptom of various diseases, such as glanders, purpura, malignant catarrhal fever. It may also be induced by injuries, rupture of the small nasal blood-vessels during severe exertion, sneezing, or it may arise from the presence of a polypus. When arising spontaneously or from injury, it must be arrested by the application of cold astringents or by plugging the nostril with cotton wool or tow. Percussion applied to the nasal region and sinuses of the head yield in the young horse but an indistinct resonance; the sound increases with age as the sinuses undergo change. The presence of pus, tumours, or coagula deadens the sound. If there be an extensive accumulation of pus, or a large tumour in the sinuses, the sound is completely deadened, the bone is painful to the touch, and often bulging in appearance.

#### AUSCULTATION OF THE LARYNX—LARYNGEAL SOUNDS IN HEALTH AND DISEASE.

In health there is a faint to-and-fro respiratory sound. In disease the laryngeal sounds are varied and important; they consist of grunting, whistling, coughing, roaring, and trumpeting. Laryngeal sounds, with the exception of "grunting," constitute unsoundness. If they be of a temporary nature, and due to irritation or tumefaction of the mucous membrane, pressure of abscesses or tumours of a removeable nature, the unsoundness may pass away; but if of a permanent character, no matter how trifling they be, they distinctly indicate unsoundness, not only interfering with the usefulness of the horse for the time being, but generally having a tendency to increase in intensity with age, and often causing a horse to become unserviceable. I do not mean to say that a whistler or roarer is not fit for work, but I assert that the infirmity is a drawback, and an animal so affected is worth less money in the market.

1. *Grunting*.—If a horse, when struck at or suddenly moved, emits, during expiration, a grunting sound, it is called a "grunter." Such a sound may or may not have any connection with disease of the larynx. A horse will grunt with pain when

suffering from pleurisy, pleurodynia, and other diseases. Some horses habitually grunt when struck at or moved suddenly. A great number of cart-horses are so affected, and big horses of all breeds are very apt to be grunTERS, whilst they may be quite sound in their wind. Horses with heavy jaws and ill-set necks often emit this sound; and again any horse may do it if it has been fed for a time with bulky food. The sound is always to be regarded with suspicion, and the animal further tried for its wind, as it generally accompanies roaring or whistling. If the grunter, however, stands the tests used to detect roaring, without making any noise in its breathing, it may be considered sound. Grunting is often symptomatic of occult glanders.

2. *Whistling*.—This sound is of two kinds, a soft or moist and a dry or hard whistle.

*Soft whistling* is due to acute laryngitis, when a considerable increase of mucus has taken place, and when the mucous membrane is temporarily swollen. When due to the presence of mucus it partakes more of a wheezing sound, and is generally diminished when the animal coughs. The sound arising from tumefaction of the laryngeal mucous membrane is greater during the inspiratory than the expiratory act. It is very similar in character to the dry sound to be described; but in addition to the sound, symptoms of catarrh, fever, or acute irritation of the larynx are present. This sound indicates a temporary unsoundness, and in many instances it is unsafe to pronounce a decided opinion for several days or even weeks after the subsidence of other symptoms. In such instances the mucous membrane has undergone some degree of thickening; there is relaxation of the vocal cords, a want of tone in the larynx generally, that require some time to overcome. It is impossible to pass an animal of this kind as perfect, and it would be unwise to give a decided opinion until all abnormal sound has disappeared.

*Dry whistling*.—This sound has been generally looked upon as a modification of roaring. I am, however, of opinion that whistling and roaring are due to different pathological conditions of the larynx, and that they may exist independently of each other; that roaring does not always terminate in whistling, nor whistling in roaring.

Whistling, like roaring, is a sound emitted more particularly during inspiration, and is due to a diminution of the calibre of the larynx, or sometimes trachea, owing to a permanent thicken-

ing of the mucous membrane, distortion of the neck by tight reining, the presence of an immoveable tumour in the trachea, or by any cause which diminishes the area of the passage of the air to and from the lungs. Whistling, although loudest during the inspiratory movement, is by no means always absent during the expiratory act; careful auscultation is, however, necessary to detect it. If entirely absent during expiration, the seat of the lesion causing it is to be looked for in the larynx or upper part of the windpipe. Whistling, like roaring, is often traceable to hereditary taint, and is an unsoundness.

3. *Roaring*.—This symptom of disease consists in a loud unnatural sound emitted during the inspiratory act. Roaring is sometimes symptomatic of acute laryngitis, and then only indicates a condition of temporary unsoundness; again it may be due to a poison (see *Lathyrus sativus* and Lead).

The cause of roaring is, however, generally found to be due in the majority of cases to atrophy and fatty degeneration of the muscles of the larynx. The origin of the paralysis is involved in some mystery; but it is generally accepted by veterinarians that, inasmuch as it is generally confined to the left laryngeal muscles, the explanation is to be found in the fact that the recurrent or ascending laryngeal nerve on the left side leaves the pneumogastric further back than on the right, and winds round the posterior aorta; whereas the right is given off opposite the first rib, winding round the dorsal artery, and consequently the left nerve is more apt to be implicated in any disease of the chest than the right. This explanation is unsatisfactory on many accounts. 1st. Roaring is not a common sequel to pulmonary disease: 2d. It is not an accompaniment of thoracic disease, which would most certainly be the case if the nerve were implicated in an inflammatory affection: 3d. Many roarsers whose history has been known from their birth have never suffered from any chest affection, nor indeed from any disease beyond a common cold, and sometimes not even from that slight ailment: 4th. Mares and ponies are less often affected with roaring than large geldings and stallions, but are quite as susceptible to chest diseases; and lastly, dissections have failed to discover any change in the nerve trunk itself, although the animals dissected have been confirmed roarsers for years previous to their death.



It is very true that division or ligature of either of the recurrences will be succeeded by roaring, and tumours involving the recurrences may cause the same symptom; and Dr. Warburton Begbie says that "no more interesting variety of local paralysis exists than that which is due to the interference with the recurrent or motor laryngeal nerve, produced by an aneurism of the arch of the aorta, or by a cancerous mediastinal tumour. Well-marked atrophy of the muscles of one side of the larynx has, under such circumstances, been found." But in the form of disease involving the laryngeal muscles, which commonly causes roaring, there is no change in the nerve itself, nor can the loss of motor power be traced to pressure upon the nerve trunk by any tumour, aneurism, or adventitious substance.

The disease of the muscles is, however, essentially nervous in its origin, and may, I think, be classified as a form of wasting palsy—paralysis atrophica—originating in the laryngeal muscles themselves. Wasting palsy is defined by Dr. William Roberts, in Reynolds' *System of Medicine*, to be "an atrophic degeneration of certain groups of muscles, independent of any antecedent loss of mobility, or of any metallic poisoning."

I have already stated that the atrophic change is usually seen on the left side of the larynx. It, however, by no means follows that the muscles of the right side are entirely free from disease; indeed, in many instances, they distinctly partake of the atrophic change, though to a less extent.

Why the change is greater on the left than on the right side, is one of those things for which no more satisfactory explanation can be given, than why the ulcers of glanders are oftener seen in the left than in the right nostril.

Roaring is generally gradually developed. At first, the sound may be intermitting, and days or even weeks may elapse during which the animal may make no noise, although put to severe exertion, as if the muscles had, at the time the sound was emitted, been debilitated from some ephemeral disturbance of nutrition. As the loss of muscular substance progresses, there is a corresponding and permanent loss of power, and what at first was intermitting is now a permanent infirmity. This intermission of the sound is not, however, the ordinary method by which the disease manifests itself. More commonly the noise or roar, slight at first, gradually, but often very slowly, increases in

intensity, thus marking or indicating the progress of the muscular change, and inability to dilate the laryngeal opening.

*Causes.*—Putting aside all exciting causes of alteration of the air passages, to be hereafter mentioned as causes of roaring, I think that laryngeal muscular atrophy is due to hereditary predisposition; and it is a well-known fact that certain breeds, the produce of certain sires, are nearly all roarers. I have repeatedly observed this, not only in racers, but in other breeds of horses. Indeed, I know one breed of Clydesdales which are nearly all roarers, both mares and horses. Horses and geldings are, however, more liable to become roarers than mares, which seldom, except where the hereditary taint is very strong, become roarers. Small ponies are scarcely ever affected, although they are subject to colds, laryngitis, and pulmonary diseases.

A horse predisposed to become a roarer generally suffers from laryngeal irritation from trivial causes, and usually the infirmity appears after repeated attacks of cold and sore throat.

*Other causes of Roaring.*—Concluding that the majority only of cases of roaring are due to the condition described, it is necessary to mention that the sound may arise from other and incidental conditions which, although not so frequent, are quite as important.

Roaring may be due to disease of the nose, nasal polypi, depression of the nasal bones from previous fracture (see *Principles and Practice of Veterinary Surgery*), osseous tumours in the nostrils; closing of one nasal chamber by false membrane or disease of the bones; tumours on the posterior nares, called “bellones” by horse-coupers, falling into the glottidean opening, causing intermitting roaring; constriction of the trachea; tumours in the thoracic cavity; distension of the guttural pouches; disease of the pharyngeal and parotidean lymphatic and salivary glands; tight reining; fractures of the tracheal rings, or any cause of distortion of the larynx.

In addition to the sound emitted during inspiration, the roarer generally has a cough which is diagnostic, being a loud, harsh, dry sound, half roar, half cough; and the generality of roarers are also grunters. It will also be found that the sensibility of the larynx is diminished in confirmed roarers, and that consequently it is difficult to make them cough in the ordinary way by pressing the larynx. In testing a horse for its wind, it is usually the

practice with some to place it against a wall, and threaten it with a whip; if it grunts, it is further tested; if not, it is merely made to cough by pressing the larynx, and if the cough has a healthy sound the animal is generally passed sound.

This plan is not always satisfactory, and is, to my mind, most reprehensible; as, if a horse be at an auction sale, and has its larynx pressed by forty or fifty horny handed coachmen and grooms, the chances of an attack of laryngitis are very great,—the better way is to have the animal galloped; or if a cart-horse, to move a heavy load some little distance, when, if it be a roarer, it is sure to make a noise.

*Treatment of Roaring.*—If the sound can be traced to any removeable cause, the practitioner knows what to do, but the removal of the causes of the form of roaring generally met with is, however, a matter of great importance. Many experiments have been tried; in some rare instances the application of blisters, or the actual cautery, to the skin of the laryngeal region, has succeeded, not only in arresting the progress of the atrophic change in the muscles, but in materially improving their strength and tone; but in order that this may prove effectual, the animal is to be treated in the very earliest stages of the disease. In addition to “firing,” I have only to suggest that the chlorate of potash is worthy of trial, as it is found to have some power in arresting fatty degeneration.

In very bad roaring the sound may be modified by pads attached to the bridle and fitted over the false nostril. These pads regulate the quantity of air taken in at such inspiration; for it is observable that when a roarer is pushed in its work or paces, that the nostrils dilate greatly, thus admitting a large volume of air, which by its weight and pressure causes a further falling in of the arytenoid cartilage and an increased constriction of the laryngeal opening. If this method be inefficient, tracheotomy is to be performed, and a tube kept in the trachea for the remainder of life. Excision of the left arytenoid cartilage (presuming that the left muscles are atrophied) has been suggested by Günther of Hanover, and has, I believe, been more or less successfully carried out. The operation is to be performed by making an incision through the skin into the superior part of the trachea, the collapsed cartilage seized by forceps or tenacula, and excised by means of a pair of scissors.

It will be apparent that a bad roarer, no matter what the means may be by which the sound is modified, is only useful for slow work ; and lastly, I have always observed that confirmed roarers become bad thrivers, and often succumb to trivial diseases.

Roaring being due to a progressive disease, it is necessary that the veterinarian should reject as unsound every animal which makes the slightest roaring or whistling sound in its breathing.

There are two terms in common use by horsemen—namely, “high-blowing” and “thick wind.”

*High-blowing* is a respiratory sound, caused by the nostrils ; it is principally an expiratory sound, caused by a *fluffing* or *flirting* of the nostrils—almost a snorting sound. It occurs in horses which are fresh and fit, and usually just when brought out of the stable, particularly on a frosty morning, or when joining the company of other horses, or when excited whilst hounds are in covert and waiting for a view-hallo. It is not an unsoundness, and ceases as soon as the horse *settles down to his work*.

*Thick wind* refers to a variety of lung sounds, or, rather, to a peculiarity of breathing due to an abnormal condition of some portion of a lung. A thick-winded horse is one which shows signs of distress and has a laboured method of breathing when subjected to very little exertion. It may be due to lack of condition, grossness of body, to plethora, to a narrowing of smaller bronchial tubes due to a chronic bronchitis, or to a patch of consolidated lung after an ordinary pneumonia, or due to some specific disease such as glanders. It is an unsoundness which may or may not be permanent.

Many people confuse the terms *thick wind* and *broken wind* ; at the same time it is not at all unusual to find that a broken-winded horse is also a thick-winded horse, but it must be remembered that a *thick-winded* horse is not necessarily a *broken-winded* one.

#### COUGH AS INDICATIVE OF DISEASE.

Cough is the sound produced in the larynx by the violent expulsion of air from the lungs, and is symptomatic of various diseases. Cough is divided into dry and moist.

*Dry cough* is divided into short, hacking, hollow, broken-winded, and spasmodic. The dry cough is symptomatic of irritation and dryness of the respiratory mucous membrane. In the early stages of laryngitis it is loud and long, becoming afterwards rasping and then moist. In chronic disease of the larynx it is loud, roaring, and often hollow. In the early stages of bronchitis it has a hollow metallic sound; it afterwards becomes moist, and is more or less painful throughout the disease. In pneumonia the cough is short, seems as if proceeding from a solid organ, and is accompanied in the later stages by a rusty, tenacious expectoration.

*The cough of pleurisy* is dry throughout, is painful, hacking, sometimes as if cut in two, the animal being seemingly afraid to complete the act by one effort.

*The broken-winded cough* is at first spasmodic, becoming, as the disease advances, feeble, short, and single, the animal being unable to relieve itself by the action of the chest and lungs; hence the suppressed cough becomes diagnostic.

*The hollow cough*, a sepulchral sound, varying in intensity, indicative of chronic disease, and on this account is termed a "chronic cough."

*The moist coughs* indicate an inflamed and humid condition of the respiratory mucous membrane.

There are various other kinds of coughs associated with diseases of the heart, digestive organs, and the process of dentition; these are valuable as aids to diagnosis, when studied in connection with other symptoms. They are mostly dry coughs.

Horses with narrow, shallow chests, weak loins, and long legs are very generally predisposed to cough from very trivial causes.

#### TRACHEAL SOUNDS.

Except at its entrance into the chest, the trachea yields no sound. At this point, however, a blowing sound—tracheo-bronchial respiration—can be heard, more prolonged during the expiratory act. In disease this may be increased. When the bronchi contain much mucus or other fluid, a moist rattle—mucous rale—may be heard, accompanied by a wheezing, gurgling, or spumous or frothy sound.

## THORACIC SOUNDS.

The thoracic sounds are divided into pulmonary, pleural, and cardiac. The pulmonary sounds are of two kinds, namely, the vesicular and the bronchial sounds.

1. *Vesicular*, also called the respiratory murmur, is heard during both inspiration and expiration. During the inspiratory act it is a soft diffused murmur of a gentle breezy character: slightly harsher and more hollow during expiration, and not above one-fourth the length of that during inspiration. It is caused by the entrance and expulsion of air to and from the terminal portions of the bronchi and air sacs. The intensity of this sound is increased with exercise or other causes of quickened respiratory movements, as fever; is louder in chests thinly clothed with flesh; stronger in the young than in adults, hence, when strong, it is called "puerile"; it is also louder when the stomach is empty. Puerile respiration depends upon the smaller size of the air vesicles, and the greater elasticity of the lung tissue; in the very old the murmur is scarcely preceptible, and is called "senile." Senile respiration may also result from slow breathing, or any cause which obstructs the entrance of air into the lungs. If either modification is present to the same extent in the same region of both sides of the chest, they indicate no other condition than the natural ones above indicated; but if the murmur is puerile on one side and senile or absent on the other, the first indicates a compensating action of that lung, supplementary to diminished action in the other. The vesicular murmur is best heard in the superior portion of the lower third of the chest, from behind the elbow and shoulder to about the ninth rib, whence it diminishes in force, and is altogether lost over the sixteenth. In the inferior part of the chest it diminishes at the seventh, and is lost at the tenth rib. On the left side it is mixed in the lower and anterior regions, immediately behind the shoulder, with the sounds of the heart.

2. *The Bronchial or Tubal sound* resembles the blowing of air quickly through a tube; it is higher in pitch than the vesicular murmur, and more rapidly evolved. It is nearly as prolonged during inspiration as expiration, with a distinct interval between the two. This sound is distinct over both the middle and upper

thirds of the chest; loudest immediately behind the scapula and caput magnum, or the nearer we can approach the ear to the bifurcation of the trachea and larger bronchial tubes; diminishing in intensity to the twelfth rib, where it is superseded by the vesicular, and entirely lost at about the seventeenth rib. It also diminishes in intensity as it approaches the upper part of the lower third of the chest, where it becomes lost in the vesicular sound. The tubal sound becomes diagnostic of disease when it is heard in the inferior portions of the chest.

In the ox, the bronchial sound is heard much lower down in the right side, owing to the large tube which passes to the anterior lobe of the right lung.

The true thoracic sounds are very often complicated with abdominal rumblings or gurglings, due to the movement of food, fluids, and gases within the alimentary canal; these are loudest in the posterior part of the chest, are irregular in their occurrence, and bear no relationship to the respiratory movements. A little practice will soon enable the student to distinguish these from the respiratory sound.

*Percussion.*—The sound obtained by percussing the healthy chest is loudest over those parts where the bronchial sounds are best heard, except indeed in those parts most thickly clad with muscular tissue. On the left side the resonance is very clear immediately behind the shoulder to the twelfth or thirteenth rib, where it gradually diminishes. If the parts are here struck forcibly, the intestinal resonance may be induced.

Mr. Percivall has pointed out what may be easily verified in practice, that the sound along the right superior region grows louder from the posterior border of the shoulder to the last rib, whilst on the left it gradually diminishes on the same line. This increase of sound on the right side is due to a resonance from the arch of the colon. If the blows are light, this sound is not brought out, and when heavy, it is more of a tympanitic character, resembling that of an emphysematous lung. Along the middle region louder sounds are elicited than either above or below, more particularly between the fifth, sixth, seventh, and eighth ribs, from whence it diminishes to the fifteenth, and then becomes tympanitic or abdominal on the left, and dull on the right side, owing to the opposition of the liver. In the inferior part of the chest the sound is weak, but clear,

from the fifth to the eighth rib on the right side, where it becomes dull, responding to the liver; whilst on the left side the resonance is almost absent over the fifth, sixth, and seventh ribs, opposite to the heart; it becomes clearer over the eighth, behind which it loses its intensity, and is lost at about the thirteenth rib.

#### MORBID SOUNDS.

The morbid sounds indicative of diseases of the respiratory apparatus elicited by auscultation may be divided into bronchial, pulmonary, and pleural.

#### BRONCHIAL SOUNDS.

These are of two kinds, namely, 1st. Dry; and 2d. Moist.

1. *Dry sounds* are subdivided into large and small, or rhonchus and sibilus.

(a.) *Rhonchus*, a hoarse, sonorous murmur, sometimes of a humming, cooing, or snoring character, compared to the bass note of a violin or cooing of a pigeon, especially marked during expiration, but coexistent with both movements, and due to a narrowing of some part of the larger bronchial tubes. It may be heard at the front of the chest very plainly, as well as behind the shoulder. It is sometimes caused by portions of viscid mucus adhering to and obstructing the larger tubes, acting as vibrating tongues as the air passes by them. If the adherent mucus be removed by coughing, the sound may disappear for a time, and then reappear; when not removed by coughing, it may be due to tumefaction of the bronchial mucous membrane; and if permanent in a given spot, it may arise from the pressure of a tumour or other cause which may flatten an air-tube. Rhonchus essentially belongs to the larger tubes, denoting partial narrowing; is a dry sound, and, if uncomplicated, indicates a condition of no great danger. It is also called vibration, sonorous rhonchus, and sonorous rale, and is generally associated with bronchitis.

(b.) *Sibilus, Sibilant Rale, Sibilant Rhonchus*.—A high-pitched whistling, hissing, clicking, wheezing sound of variable intensity and duration, coexistent with both respiratory movements, but more especially marked during inspiration, arising from tume-



faction or accumulation of viscid mucus in the small bronchial tubes. I once heard a dairyman call this a "chaining sound," from its resemblance to rustling of an iron chain. It is associated with bronchitis situated in the smaller tubes, and is best heard over those regions where the vesicular murmur is most audible in health. It indicates greater danger than rhonchus, and if present over a large surface of both sides, a condition of great gravity.

2. *Moist sounds*.—The dry, bronchial sounds are succeeded in bronchitis by moist ones, termed rales, rattles, or bubbling sounds.

(a.) *Mucous Rhonchus or Rale*.—The bursting of bubbles of some size, unequal and varying in number, modified by coughing and expectoration, coexisting with both respiratory movements. It is due to the bubbling of air through liquid—mucus, blood, or pus—in bronchial tubes the size of a crow quill, heard in those regions where the tubal sound is most apparent in health. Succeeding dry rhonchus, it indicates the moist stage of bronchitis.

(b.) *Small bubbling Rhonchus or Rattle, subcrepitant or submucous Rales or Rhonchi*, succeeding sibilus, coexistent with both movements, but loudest during inspiration, and due to the bubbling of air through a more or less viscid fluid in the minute bronchial tubes at their peripheral distribution; heard in bronchitis affecting the smaller tubes, and during the resolution of pneumonia.

(c.) *Gurgling Rattles, cavernous Rhonchi or Rales*.—The bursting of bubbles, obviously of a large size, with a hollow, gurgling sound, or a metallic sound if the bubbles be small, coexisting with both respiratory movements, is associated with vomicae or excavations from tubercle, dilatation of bronchi, pus in pleura, with a bronchial fistula and deliquescence of gangrenous lung structure.

#### PULMONARY SOUNDS.

1. *Crepitations*.—Crepitant rale or rhonchus; compared to the sound produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear, or to the crackling of salt when scattered over hot coals. It is heard during inspiration only, and indicates the primary stage of pneumonia. "Pro-

bably due to the sudden expansion of delicate tissue, altered in its physical properties by the inflammatory state, and which probably undergoes minute ruptures.”—(AITKEN.) This sound is best heard in the lower third of the chest, replacing the vesicular murmur. After continuing for a short period it may disappear, and the vesicular murmur return, indicating the resolution of the inflammation. Usually, however, the crepitation becomes fainter and fainter, and is substituted by—

2. *Tubal or Bronchial sound.*—The tubal or bronchial sound, when heard over the inferior portions of the thorax, indicates some degree of consolidation or hepatization of the lung tissue. It shows, in fact, that the minuter ramifications of the bronchi and air vesicles have become impervious to air, by the pressure of an exudate within and upon their walls, and that the larger tubes of the part are still pervious. In health, both vesicular and tubal sounds are emitted by all parts of the lung tissue, but can be detected by auscultation in certain parts only. For example, the tubal sound is so loud in the upper and anterior parts of the chest, as to mask or hide the vesicular murmur to a great extent; and, conversely, the vesicular murmur being loudest at the inferior portions, masks or renders inaudible the tubal or bronchial sound. When, however, those portions of the lungs, namely, the minute tubes and air vesicles, are rendered impervious—the larger sized tubes still remaining pervious—it naturally follows that the sound emitted by air passing in and out of the larger tubes now becomes audible.

In many instances, however, the tubal sound continues for a short time only, and is succeeded by—

3. *Absence of sound.*—When this occurs it indicates that the exudation is excessive in quantity, that the larger as well as the smaller tubes have been rendered impervious, or that effusion has taken place into the cavity of the thorax.

Consolidation of the lung tissue, and the presence of fluid in the thoracic cavity, can be recognised the one from the other—(1.) By the previous character of the sounds; and (2.) By the manner in which the absence is marked. In hydrothorax the termination of the area of absence of sound is sharply defined superiorly; above the “water line” the pulmonary sounds are generally exaggerated. In consolidation the area of absence of sound has no sharply defined limit; at the lower parts of

the chest, and for some inches upwards, there may be no sound at all; then faint tubal sounds are heard, which increase in intensity as the ear is moved upwards along the thoracic walls. Now and then a blowing sound is heard in consolidation of the lungs, confined as it were to the superficies of the lung, indicating hepatization of the deeper-seated portions of the organ, leaving the subpleural parts still pervious. My experience leads me to the conclusion that this superficial blowing sound is a bad symptom, indicating a creeping pneumonic inflammation, having a tendency to a fatal issue.

4. *Secondary crepitations*.—Consolidation, as above described, is succeeded by the breaking up and absorption of the exudate. This process is marked by the advent of crepitations of a bubbling nature, slowly evolved, few in number, dissimilar and irregular in occurrence, more audible during inspiration than during expiration; they mark the resolution of pneumonia, and are succeeded by the natural sounds more or less modified. Such are, then, the sounds of pneumonia. They are subject to modifications, and to be variously mixed one with the other during the various phases of the disease.

#### PLEURAL SOUNDS.

1. *Friction sound*.—A grazing, rubbing, grating, creaking, irregularly jerking, superficial noise, heard more particularly in inspiration, or in both respiratory acts. It results from the rubbing together of the two opposed surfaces of the pleura, chiefly heard at the lower part of the chest, where the pulmonary organs have the greatest freedom, and is indicative of dryness of the serous surfaces, or any cause of roughness upon them. This sound is succeeded, when hydrothorax occurs, by—

2. *Absence of sound* in the inferior part of the thorax, reaching to a certain line, above which the respiratory sounds are heard.

As the fluid accumulates, the absence of sound ascends. In some cases of hydrothorax one or two other sounds are heard, namely, metallic tinkling, and a gurgling or splashing sound. These sounds are only heard when gas or air and fluid coexist in the pleural cavity. The metallic tinkles may occur from drops of fluid, imprisoned in false membranes, falling from the roof of the pleural cavity into the liquid beneath, or may result

from the bursting of bubbles of air or gas on the surface of the liquid.

In the horse the pleural sounds are less distinct than in the ox and dog.

The sounds termed bronchophony, pectoriloquy, ægophony, and amphoric resonance, produced by modifications of the voice during articulation, do not apply to any morbid sounds heard in the lower animals.

#### CARDIAC SOUNDS.

If the ear or stethoscope be applied to the lower part of the shoulder or arm, or if the left (near) fore leg be extended, and the humerus pulled forward as far as possible, the sounds of the heart can be made out.

The sounds of the heart in health are two in number: first, a longish, dull sound, then a sharp sound, succeeded by a pause, and then the recurrence of the sounds in a regular and uniform manner, provided the animal is not excited nor in any way disturbed. The first sound coincides with the hardening of the ventricles, the complete closure of the auriculo-ventricular valves, and the opening of the arterial orifices; is caused principally by the sudden distension of the ventricles, and is of the same nature as the noise made by all muscles in contracting against a resistance.

The second sound is undoubtedly due to the sudden closure of the sigmoid (semilunar) valves at the conclusion of the ventricular contraction. This has been proved by experiments on living animals, the sound being destroyed by hooking back the semilunar valves.

The impulse of the heart may be felt by placing the hand directly upon the left side of the chest, immediately behind the elbow. It is very distinct in flat-sided and lean animals, less distinct, or even entirely absent, in round-chested and fat ones. Diminished impulse, when not due to the above cause, indicates feebleness of the heart's action from disease, as fatty degeneration of its muscular tissue; hydrops pericardii, when the apex may be prevented by the effusion from coming into contact with the thoracic wall; or it may arise from weakness of the system generally, attenuation of the cardiac walls, and dilatation

of the cavities. Emphysema of the lungs may also diminish the impulse, the enlarged lung overlapping the heart; and adhesions of the pericardium to the pleuræ of opposite sides may bind down the heart so that the impulse will not be felt.

The impulse of the heart is temporarily increased by any cause of excitement, fear, exercise, fever, or pain. When excessive it is called palpitation.

Increased impulse, when not traced to the above-named causes, or when it is easily induced, may depend upon organic disease of the heart. It is stronger than natural in hypertrophy of the cardiac walls, and particularly so if such hypertrophy is associated with dilatation. It may, in such cases, be slow, gradual, and double, and this kind of impulse is due to no other condition of the heart; indeed it is one of those few symptoms which throw light upon the condition of parts within, for, notwithstanding much observation carefully recorded, we are bound to confess that, unless cardiac diseases be aggravated, we are unable, either by the character of the sounds or impulse, to diagnose them with that certainty arrived at by those who practise on the human patient.

#### MORBID SOUNDS.

1. A to-and-fro friction murmur, synchronous with the heart's movements, indicates pericarditis or pericardial effusion.

2. A bellows murmur with the first sound indicates mitral insufficiency; stricture of aortic orifice; disease of the aortic valves, or deposits on the ventricular surface of the mitral valves; or it may depend upon an altered condition of the blood itself, as in anæmia, in which case it resembles a churning sound, heard also in the large veins and arteries. These anæmic murmurs vary with the condition of the blood. Sometimes there is a continuous hum heard at the base of the jugulars, and to which the French have applied the term "*bruit de diable*."

3. A bellows murmur with the second sound indicates aortic insufficiency; roughened auricular surface of the mitral valves, or mitral obstruction. The most common sound heard in the horse is a murmur masking or hiding the second sound, and indicating semilunar insufficiency. When the murmur is double, occupying the periods of both cardiac sounds, it indicates mitral obstruction and insufficiency.

The sounds of the heart may be seemingly intensified by consolidation of the lungs and by hydrothorax, and diminished by emphysema of the lungs. In some instances of consolidation the sounds may be more distinctly heard on the right than on the left side: this indicates hepatization of the right lung, with compensating dilatation of the tubes of the left one.

Metallic tinkling, coincident with the cardiac impulse, is sometimes associated with the booming sounds of endocarditis.

#### PERCUSSION IN DISEASE.

Supplementary to auscultation, percussion is a valuable aid to diagnosis, more especially of pleural and pulmonary diseases.

1. Increased resonance indicates dilatation of the bronchial tubes and air vesicles; if partial or confined to one side only, and if associated with dulness of the opposite side, increased resonance may merely indicate a compensatory respiratory effort. In such a case the respiratory murmur will also be increased. Augmented resonance is, however, generally associated with decrease or absence of the true respiratory sounds, and accompanied with wheezing, creaking, crackling crepitations, indicative of pulmonary emphysema, as in broken wind, rupture of the air cells, and chronic bronchitis.

2. Diminished resonance indicates consolidation of the lungs, hydrothorax, pleural exudations, tubercles, congestion of the lungs, pulmonary apoplexy, &c. When confined to the inferior parts of the chest, replaced abruptly superiorly by increased resonance, it is diagnostic of hydrothorax. When the dulness gradually disappears, it is indicative of hepatization of the lung tissue, being greatest where the consolidation is most complete.

It is stated that enlargement of the heart is indicated by an increased area of cardiac dulness; for my own part, I have never been able to make this out in the horse, but have done so several times in cattle affected with traumatic pericarditis, when the deposit on the heart has been very considerable.

## CHAPTER LVIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (I.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

#### CATARRH AND LARYNGITIS.

##### CATARRH.

*Synonyms.*—Common cold, coryza, a defluxion or running at the nose, and is witnessed in the horse, ox, sheep, and other animals.

*Symptoms.*—Catarrh is indicated by sneezing, running from the eyes, redness and dryness of the Schneiderian membrane, succeeded by a discharge, at first thin and colourless, which soon, however, becomes turbid, yellowish-white, and profuse. It is associated with a varying degree of fever, dulness, and debility.

*Causes.*—Alternations of temperature, hot, ill-ventilated stables, exposure to wet and cold. A strong predisposition to cold exists during the process of changing the coat. Young animals, more especially if newly brought into warm stables, are pre-eminently liable to suffer. In some instances it is complicated with laryngitis, and in all cases, if the animal be neglected, the catarrhal inflammation is apt to spread from the nose over the whole surface of the respiratory mucous membrane. The mucous membrane of the mouth and air passages is very completely protected from the attacks of organisms by its vascularity, which facilitates the production and propulsion of numerous phagocytes whose duty it is to keep these parts free from such pathogenic organisms. If, however, the membrane is irritated by the presence of noxious vapours

such as ammonia, &c., which are the result of bad ventilation, this phagocytosis will be to a certain extent inhibited. The result will be that the organisms already mentioned will obtain a growing surface in the nasal chambers and pharynx, and the animal has what is known as catarrh. The organisms chiefly concerned are staphylococci and diplococci, but possibly streptococci, which may be the streptococcus of strangles. One can easily understand how essential it is that anything which is likely to interfere with the health of the animal should be carefully avoided, as it is these debilitating causes that lower an animal's resistance and render it susceptible to organisms which under ordinary conditions would not be pathogenic.

*Treatment.*—This is a very simple matter if adopted in time, and consists in placing the animal in a well-ventilated loose box, clothing the body if the weather be cold, feeding upon bran mashes, boiled linseed, and a small quantity of good hay for a few days.

In the early stages the nasal irritation will be much relieved by making the animal inhale steam, and this is best done by holding its head over a bucketful of hot water, and stirring the water with a wisp of hay. A few doses of nitrate of potash are beneficial in the early stages as a febrifuge and diuretic; if the supervening debility be extreme, tonics and good food are to be prescribed. Some writers recommend that a purgative should be given: this is a practice which cannot be too strongly condemned, as in all catarrhal affections there is a tendency to the ..... spread of the irritation, and a purgative may cause a fatal inflammation of the mucous membrane of the digestive organs; but, provided that the effect of such treatment is not immediately fatal, a simple disorder is rendered complicated and serious, and, should the animal ultimately recover, the convalescence is much more prolonged and difficult. Any undue constipation of the bowels is best combated by laxative food, enemas of warm water, and perhaps eight or ten ounces of linseed oil. If the cough be troublesome the throat may be stimulated or lightly blistered. For the results of catarrh, see *Principles and Practice of Veterinary Surgery*, page 517, *et seq.*



## LARYNGITIS.

*Acute Laryngitis.*—An inflammation of the lining membrane of the larynx, indicated by difficult breathing, discharge from the nose, and febrile disturbance.

*Pathology and Symptoms.*—Laryngitis is not an uncommon nor yet an unimportant disorder, sometimes killing quickly, and at all times a dangerous disease. The gravity of acute laryngitis depends upon the nature, character, and extent of the inflammation. In acute inflammation, embracing the epiglottis and rima glottidis, there is such a rapid and extensive effusion into the submucous tissue, and formation of mucus upon the free surface of the mucous membrane, as to cause almost a total obliteration of the glottal opening, and the death of the animal from suffocation. This rapid effusion into the submucous tissue and swelling of the membranes constitute what is termed “cedema glottidis,” and it is to this rapid effusion that the danger to life is due. The respirations become suddenly difficult, the inspiration being particularly prolonged, and attended with a peculiar harsh sound, succeeded by a short expiratory movement, sometimes, but very rarely, attended by a hoarseness. The animal’s nose is protruded, the superior respiratory passages being thus made to approach as near a straight line as possible; the eyes prominent, conjunctivæ red and highly injected, with abundant flow of tears. There is a peculiarly anxious and distressed expression of the face, the alæ of the nostrils are dilated, the nasal chambers are reddened, there is a hoarse rasping cough, sweats bedew the body, the legs and ears are cold, the latter often drooping; the animal manifests its distress by frequently stamping with its feet—the fore ones particularly. The slightest excitement aggravates all these symptoms; the pulse, which may at first be hard and full, soon becomes rapid and indistinct—fulness generally remaining; the visible mucous membranes now assume a livid appearance from non-oxidation of the blood; prostration of strength becomes extreme; the animal staggers, finally falls, and dies after a few struggles. The above is a description of by no means a common, but of an aggravated form of acute laryngitis. In many cases the symptoms are much less severe, but they partake of the general character of those above

described. There is generally a discharge from the nose, even in the early stages, and the act of deglutition is performed with great difficulty. In some instances this is due to the inflammation extending to the pharynx; in others, however, there seems to be no real pharyngeal complication, for if tracheotomy be performed, the difficulty in swallowing is immediately removed. It appears to me that the difficulty in swallowing is due to the momentary pressure of any liquid or solid upon the inflamed epiglottis, causing increased interruption to the ingress of air. Restore the freedom of breathing by the operation of tracheotomy, and the passage of fluids or solids from the fauces into the pharynx no longer prevents the free ingress of air, and hence the swallowing again becomes easy. In those cases, however, in which the difficulty in swallowing persists, the pharynx is inflamed, and the food is returned into the nose, tinged the mucus with its own colour.

The causes are the same as those of common cold.

*Treatment.*—In an aggravated case this must be prompt. Inhalation of steam, and hot fomentations to the throat, may be tried for a short time, but if the distress is not speedily relieved, tracheotomy is to be resorted to. In the milder cases, inhalations, fomentations to the throat, succeeded by blisters, with febrifuges; light, soft diet, warm clothing, comfort, and pure air, constitute the necessary treatment.

It may be observed that when deglutition is difficult, all medicines should be given in the animal's food or water, the latter being abundantly supplied, as enforcement may cause violent fits of coughing, and even suffocation. Belladonna sometimes has a good effect in the earlier stages; it is best given as the extract, placed between the teeth.

This disease is always succeeded by great prostration of strength; and in order to prevent this as much as possible, milk—in conjunction with eggs beaten up, or boiled hard and powdered—should be allowed the animal to drink, alternately with water, gruel, or linseed tea. But none of these should upon any consideration be forced upon it by horning or bottling, for, as I have already stated, this is a dangerous practice, and one calculated not only to excite violent fits of coughing in all diseases of the throat, but indigestion, and disorder of the digestive apparatus in all other ailments, and thus destroy what little appetite the patient might possess.

*Sequelæ*.—Thickening of the mucous membrane, ulceration of the rima glottidis, atrophy of laryngeal muscles, and follicular growths upon the laryngeal entrance.

Thickening of the mucous membrane is best removed by a course of iodide of potassium and blisters; ulceration of the rima glottidis, by solution of nitrate of silver applied to the part by a sponge fastened to a rod. The follicular growths have been removed by the application of solution of corrosive sublimate—forty grains to the ounce of water; and to prevent the progressive atrophy of the muscles, I would recommend a trial of the chlorate of potash.

*Chronic Laryngitis*.—(See *Roaring*.)

## CHAPTER LIX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (I.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

##### INFLAMMATION OF THE RESPIRATORY ORGANS— BRONCHITIS.

*Division.*—This disease may, according to its seat, be arranged under four heads, namely, “tracheo-bronchitis,” where the lower part of the trachea and larger tubes are the main seat of the inflammation; “bronchitis proper,” where the medium-sized bronchi are the chief seats of the disease; “capillary bronchitis,” where the smaller bronchi are chiefly implicated; and catarrhal, lobular, or broncho-pneumonia, where the smallest bronchi and alveolar walls are involved in the inflammatory process. For simplicity of description I shall retain the generic term bronchitis, dividing it into acute and chronic.

The character of the inflammation, whatever part of the respiratory tract may be affected, is what is understood as catarrhal—that is, an inflammation in which, instead of an exudation rich in fibrin, there is a fluid secretion containing a large quantity of mucus and cellular elements. In this particular it differs most essentially from inflammation of the lungs, originating in the parenchyma and from pleuro-pneumonia, in which the pleural surface as well as the lung structure is involved. The exudate in these is termed “croupous” or fibrinous.

*Causes.*—Bronchitis, wherever its seat, is generally due to exposure to cold; it may supervene on an attack of ordinary catarrh, particularly if the animal be neglected, exposed to wet and cold, or kept in ill-ventilated stables. It may also arise without any premonitory catarrhal symptoms in both horses and cattle during voyages by sea, particularly if the weather be rough and stormy, and the animals battened down. During 1877 the author had the opportunity of seeing bronchitis assuming the form of suffocative catarrh, and which proved fatal to many, amongst foreign horses imported at Leith. An instructive fact

in connection with these cases was that it appeared only after rough and stormy passages; when the weather was fine no cases were observed. It also arises from the absorption of septic matters, as seen in post-parturient septic broncho-pneumonia, particularly when the foetal membranes are retained, and in the so-called corn-stalk disease in American cattle described by Billings (see p. 188), and as a result of drinking water tainted with the putrefactive products of animal matters. A similar condition is described by Poels, Holland, and Professor E. Lienaux, Brussels, as septic pleuro-pneumonia in calves, and in which they have demonstrated a mobile ovoid microbe  $\cdot 001$  mm. to  $\cdot 0015$  long by  $\cdot 0005$  mm. in breadth, identical in appearance to the one observed by Nocard, Billings, and myself (see Plate VI<sup>1</sup>).

Bronchitis, like laryngitis, may be caused by the inhalation of irritant matters, more particularly smoke—which form is usually very acute with hæmorrhagic frothy discharge, and terminates fatally—and by the accidental entrance of foreign materials, as medicines or food, into the bronchial tubes. Inflammation of the bronchial tubes arising from the latter cause usually occurs in horned cattle, often as a sequel to parturient apoplexy, in which affection the power of deglutition is in a great measure lost, and where the sensibility of the glottis is, during the comatose stage, greatly diminished or entirely absent. In such cases fluid medicines incautiously administered enter the trachea and bronchi, and these may cause immediate death by suffocation, or if not immediately fatal, induce a severe and perhaps fatal inflammation. Bronchitis is probably always the result of a bacterial invasion, usually those facultative organisms of the *Pasteurella* type. These organisms may set up the initial condition, but may not persist; still, having commenced the inflammatory process, they prepare the ground, as it were, for organisms which ordinarily would not be pathogenic, and this would account for the prolonged course of the disease.

Again, during the state of coma, semi-fluid ingesta are apt to flow into the mouth through the flaccid œsophagus, particularly if the cow lies with its head and anterior extremities lower than the posterior ones. In parturient apoplexy there is also very often during the earlier stages some extent of antiperistaltic action of the œsophagus, with eructations of gases from the rumen; along with such gases semi-fluid ingesta gain entrance

into the fauces, and owing to the paralyzed state of the glottis fall into the larynx and trachea.

Catarrh or bronchitis, from other than mechanical causes, may, particularly in cattle, if the accompanying cough be long and powerful, cause some degree of vomition. The food thus vomited, or in other words coughed up, sometimes gains entrance into the trachea, and causes a fatal issue.

Along with Mr. Borthwick, Kirkliston, I saw cases of this in a herd of Irish cattle brought to Scotland, which were suffering from bronchitis and gastric irritation from neglect and exposure.

Four of the herd became much worse than the rest, one died, and the other three were slaughtered. In all of them the bronchial tubes were filled with ingesta, ejected into the fauces during violent fits of coughing. Again, in several specimens of the lungs of American cattle slaughtered at Liverpool, supposed to be affected with pleuro-pneumonia, food was found in the bronchi. Is it not possible that during a rough voyage cattle may suffer to some extent from sea-sickness, and even vomition, and that the vomited matters may gain access into the trachea and bronchi? In others of the condemned American cattle the irritation was associated with the presence of filaria in the bronchi. Both the ingesta and the parasites were present only in a minority of the diseased lungs examined, and could therefore be only looked upon as accidental concomitants.

Food sometimes gains access into the trachea in the course of dissolution, or even after death, particularly if the rumen be rather full of moist food; it will then be found in the greatest abundance in the trachea and larger bronchi, whereas in those instances in which it has been in the tubes for some time before death, the food will often have disappeared from the larger into the smaller tubes and air cells.

I have witnessed one case of fatal bronchitis in the horse, due to the entrance of vomited ingesta into the bronchi. Some days prior to its death fifteen minims of Fleming's tincture of aconite had been administered; this brought on attempts at vomition and great distress. The animal's respiration continued very highly accelerated after the effects of the aconite had passed off, and continued until the animal died. A *post mortem* examination revealed the fact that vomition had occurred, and that the small quantity of food thus expelled had entered the larynx, and gained access to the bronchi.

## ACUTE BRONCHITIS.

*Symptoms.*—Bronchitis consists of congestion of the bronchial tissues, associated at first with dryness, narrowing, and rigidity, and subsequently moisture, dilatation, and relaxation of the tubes.

Owing to these changes, the vibrating sounds caused by the passage of air through the inflamed bronchi undergo variations, which indicate pretty clearly the dry or moist condition of the parts, or, as some term it, the dry or moist catarrh.

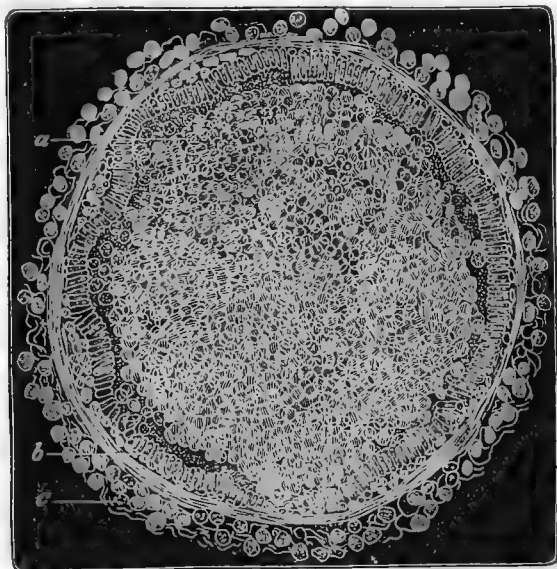


FIG. 75.—Small healthy bronchus in acute bronchitis, occluded by a plug of catarrhal secretion. —350 diam. *a*, Catarrhal plug; *b*, Epithelium lining bronchus; *c*, Surrounding adventitious coat infiltrated with cells. —(From American ox condemned at Liverpool for pleuro-pneumonia.)

As the symptoms are developed, the cough becomes hoarse, ringing, loud, and paroxysmal; the respirations are in some instances greatly accelerated, indeed out of all proportion to the pulse. For example, the pulse may be seventy or eighty per minute, and the respirations as numerous, or even more so: this indicates bronchitis affecting the smaller tubes and alveolar walls—catarrhal pneumonia—collapse of a more or less extensive area of lung structure, or even occlusion of non-inflamed bronchi and air vesicles by the gravitation into them of the catarrhal fluid, as shown in the woodcut.

Bronchitis of the larger tubes is naturally less dangerous than the other two, and only proves fatal by inducing the two above-mentioned conditions, namely, collapse and occlusion of a more or less extensive breathing surface.

Amongst the foreign horses above alluded to, it was noticed, where the discharge of muco-purulent matter was most profuse, although some of the animals seemed to recover from the febrile disturbance and accelerated breathing of the acute stage, that they succumbed in from fourteen to thirty days afterwards from gangrene of the collapsed lungs, or putrefaction of the fluid incarcerated in the bronchi and air cells; both of these conditions being expressed by fœtor of the breath, exhaustive diarrhœa, metastatic inflammations of the articulations and feet, complete loss of appetite, rapid emaciation, fluttering pulse; at first great elevation of temperature—106° F. or more; partial sweats upon the body, gasping respiration, some abdominal pain, and other signs of general septicæmia.

In no case of pure bronchitis is the breathing painful, but short and quick, the thoracic as well as the abdominal muscles being brought into full play; this distinguishes it from the breathing characteristic of pleurisy, in which the ribs are more or less fixed and the respirations abdominal. In ordinary cases of bronchitis the animal is dull, listless, sometimes semi-comatose; hangs its head; is generally thirsty; ropy saliva fills the mouth, which is hot and moist. The visible mucous membranes are injected, and present a varying degree of lividity, due to non-oxidation of the blood. The animal stands in a corner or moves listlessly about. If in a box, and the door be open, it stands with its head to the open air, from which it evidently obtains relief. The bowels are generally somewhat constipated, the fæces covered with mucus, but they easily respond to purgatives, showing that the alimentary mucous membrane participates in the irritation. The urine is high-coloured, scanty, and if examined will be found to contain urea, mucus, and colouring matter in excess, and the chlorides in diminished quantities.

As already stated, bronchitis of the larger tubes is not ordinarily a fatal disease, but when affecting the smaller bronchi and alveoli, particularly if associated with a profuse discharge of a yellowish coloured, more or less tenacious fluid, which occludes the smaller bronchi and air cells, it is the most fatal chest disease that the author is acquainted with. This tendency to gravitation of the



catarrhal fluid is explained by the fact that the columnar and ciliated epithelium are shed in the earlier stage of the attack, and take no part whatever in the after changes which ensue. It is never seen again till the signs of acute inflammation, such as distension of the vessels and œdema of the basement membrane, have passed off. Subsequently it is gradually reproduced.

The muco-purulent material thus incarcerated is driven or impacted by the ramrod-like action of the inspired air into the periphery of the smaller tubes and vesicles, and there constitutes those masses which may undergo putrefaction in the horse, causing septicæmia, as already explained, and caseous masses, mistaken for tubercle in the ox.

The physical signs of bronchitis are as follows:—Percussion returns a more or less resonant sound, but auscultation will enable the practitioner to detect the nature and extent of the bronchial inflammation. *Rhonchus*, confined to the upper and middle third of the chest, with true respiratory murmur over the lower part, will indicate inflammation of the larger and middle sized bronchial tubes, and a condition of comparatively little danger. *Sibilus*, heard at the lower parts, indicates a condition of much greater danger, and that the disease involves the smaller tubes and air vesicles. Inspiration is generally shortened, expiration prolonged, and more distinctly accompanied by the abnormal sounds. These sounds are succeeded at a later stage by moist bubbles, rattles, or rales—mucous rales. At first the discharge expelled by coughing is thick, tenacious, and gelatinous, or watery and scant. The lower animals do not, however, expectorate in the true sense of the word; some discharge issues from the nose, but the greater part of what is coughed up falls into the fauces, and is swallowed. As the disease advances, however, a profuse discharge issues from the nostrils, and the inflammation gradually subsides. The cough becomes less hoarse, more vigorous, and even more frequent than at first; but it gradually disappears, the discharge becomes again thinner, clearer, and eventually ceases.

In some instances all sounds disappear from a certain part of the lungs. This is due to occlusion of the tubes and vesicles by the catarrhal secretion, or to more or less collapse of the vesicular tissue, dependent on obstruction to the passage of air during inspiration by glutinous or inspissated mucus. This collapse is often confined to individual lobules, which are thus

condensed, heavy, indurated, and of a dark colour, and may ultimately become hepatised, atrophied, or even emphysematous.

#### PATHOLOGY AND MORBID ANATOMY.

Inflammation of the bronchial tubes, like that affecting other mucous membranes, is attended with changes in their epithelium, the secretion of the glands, and in the surrounding tissues.

It is rare to meet with a fatal case of bronchitis during its earlier stages, and but for the accidental slaughter in Liverpool of the American cattle already referred to, it would have been difficult to have given the details of the morbid anatomy.

The appearance of the lung in the earlier stage of bronchitis, with collapse, that is to say, when it is observed prior to the commencement of secondary changes or phenomena, is as follows:—There are patches over its surface that have fallen below the level of surrounding parts; sometimes these depressions measure an eighth of an inch in depth; they are of a bluish-purple colour, and variable in size. The parts around them are of a light pink hue, and are either healthy or in a more or less emphysematous condition.

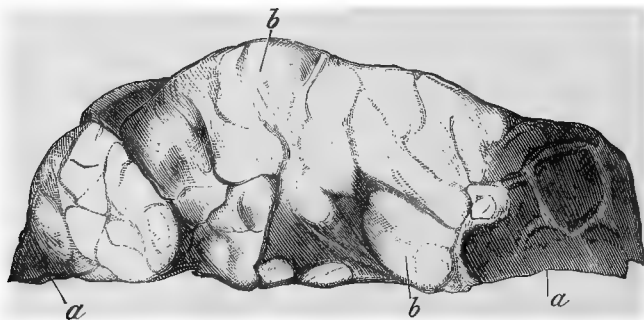


FIG. 76.—Portion of lung from American ox slaughtered at Liverpool, and showing bronchitis in the very earliest stages (*a a*, collapsed lobules from obstruction of tubes. The elevations (*b b*, non-collapsed lobules) are slightly emphysematous.

The depressions consist of certain lobules in a state of collapse arising from occlusion of their bronchial tubes by pus or other material. The collapsed portions are bluish-purple in colour; non-crepitant, and depressed, resembling foetal lungs, sinking slowly in water.

Collapse of the lung tissue—atelectasis—induces more or less

congestion and subsequent inflammation; consequently it is found that broncho-pneumonia often succeeds bronchitis, due to the absence of the expansion and contraction of the air vesicles which normally aid the pulmonary circulation, and to arrestment of the blood-flow owing to imperfect æration. This congestion is soon succeeded by effusion of serum, and the bluish-purple collapsed portions become darker in colour and less resistant in consistence. They, however, retain some degree of elasticity, for, if not too rudely pulled out, they do not tear as in pleuro-pneumonia; if cut into and exposed to the atmosphere for a few minutes, the bluish-purple colour becomes bright scarlet. It is important to bear in mind that the pneumonic process which supervenes in bronchitis is principally confined to those portions of the lungs in which collapse has taken place. Sometimes the collapse is isolated, invading but small portions of the lungs: this condition is not rarely witnessed in parasitic bronchial disease. These limited collapsed portions vary in size, are rather wedge-shaped, and have their apices towards the obstructed bronchus. The lung tissue surrounding them may be more or less congested, or it may be emphysematous, but no juice is exuded from them when cut into, as in acute pleuro-pneumonia.

Professor Gairdner was, I believe, the first to show that condensation of the vesicular substance occurs as a result of mucous or other obstruction in the air-tubes leading to the condensed portion. It is at first sight difficult to understand how incomplete obstructions of the bronchi—and these obtain much more frequently than absolutely complete occlusion—cause collapse. One would suppose that some quantity of air would gain access into the vesicles, but such is apparently not the case; and it seems that the air gradually finds its way out by the edges of the obstructing substance. The expiratory force, so long as there is air in the vesicles, constantly tends to dislodge the obstructing body by pushing it towards the wider (proximal) end of the tube, whilst the inspiratory drives it inwards towards the narrower tubes, which it effectually occludes. The entrance of air is thus more or less effectually opposed, and its exit permitted, so that ultimately the vesicles beyond become completely emptied; in fact the plug acts as a valve, allowing the air to pass in one direction, but opposing its passage in the other. Where the

obstruction is complete from the commencement, the air is absorbed.

It had been supposed by Laennec that the emphysema, or, more correctly, the over-distension with air of the parts surrounding the collapsed lobules, was due to what he thought a fact, that the act of inspiration was more powerful than that of expiration, so that though air could be drawn through the obstruction, it could not be breathed out. In consequence, it accumulated in the ultimate pulmonary vesicles, became expanded by heat, and so acted mechanically as a dilator. Dr. Gairdner, however, pointed out that expiration is a much more powerful act than inspiration, and that there is never any difficulty in causing expulsion of air, provided always there be no obstruction in the tubes. Emphysema, then, does not occur in the vesicles connected with obstructed tubes, but in those which are adjacent. When the lungs are free from disease the column of air presses equally in all the tubes and vesicles; but when one portion connected with any obstruction is collapsed, then the adjacent parts are over expanded, so as to occupy the space previously filled by the former.

At a later stage the contents of the obstructed bronchi are pushed by the weight of the descending or inspired atmosphere into the most minute bronchi, alveoli, and air vesicles, always from the centre towards the periphery, and appear as minute white points beneath the pleural surface. They are well shown in the figure.



FIG. 77.—Pleural aspect of pulmonary lobe from American ox slaughtered at Liverpool; alveoli filled with muco-purulent matter; pleural surface intact. The microscopic examination revealed broncho-pneumonia in some of the alveoli (see fig. 79); whilst others showed no traces of inflammation (see fig. 75), but were merely filled with the inhaled bronchial secretions.

On cutting into the lungs, it will be found that the large and small tubes, and sometimes the trachea, contain an amount of fluid. This condition, as well as the collapse, is limited in the majority of instances to the small or anterior lobes of the lungs, and rarely, except by extension, affects the large lobes, not only in ordinary but in mechanical bronchitis. This fact is of importance, as pleuro-pneumonia contagiosa, with which the disease under consideration has been confounded, generally commences in the larger lobes, either in their centres or towards their posterior edges.

The fluid contained in the tubes is thick, and has a yellow colour; in the trachea it is more or less frothy; and is abundant in the smaller bronchi. as shown in the figure.

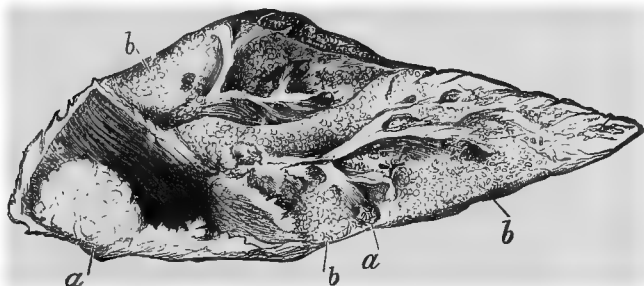


FIG. 78.—Section of portion of lung; the external aspect is shown in fig. 77. The larger (a) and smaller bronchi and air vesicles (b) filled with purulent matter.

If the lungs in this condition be squeezed, little pellets of yellow matter are pressed out: sometimes these pellets are too small to be seen by the naked eye, and require the aid of a magnifying glass. If the bronchitis be associated with catarrhal pneumonia, elevated patches will be apparent on the cut surface, having a greyish-red colour. They are soft to the touch, and if squeezed, the same muco-purulent matter exudes from them, or from a small bronchus which may happen to communicate with the particular group of vesicles implicated.

Dr. Hamilton, in his series of papers on bronchitis published in the *Practitioner* for 1879, states it is a matter of difficulty in man to get at the first change which ensues in the bronchi in acute catarrh. He has, however, been able to verify his observations by an examination along with myself of the lungs of American cattle slaughtered in the earlier stages of bronchitis;

in fact before any external signs of disease were manifested. He says—"On careful comparison, however, of many cases, we feel assured that the first deviation visible is a *relaxation and distension of the abundant plexus of blood-vessels ramifying in the inner fibrous coat*, immediately beneath the basement membrane—that is to say, of the branches of the bronchial artery. They become engorged with blood, so that on transverse section they appear like little cavities distended with blood corpuscles. In a few hours afterwards the basement membrane<sup>1</sup> becomes much more apparent than it usually is, and at the same time more clear and homogeneous, while the surface is thrown into many folds. These changes in the basement membrane are apparently due to its becoming œdematous, serous fluid being infiltrated into it from the underlying plexus of distended vessels; and we shall see that, as the acute irritation continues, this œdematous state of the basement membrane becomes more and more a well-marked feature. The next change, so far as we have been able to calculate, occurs in from twenty to thirty

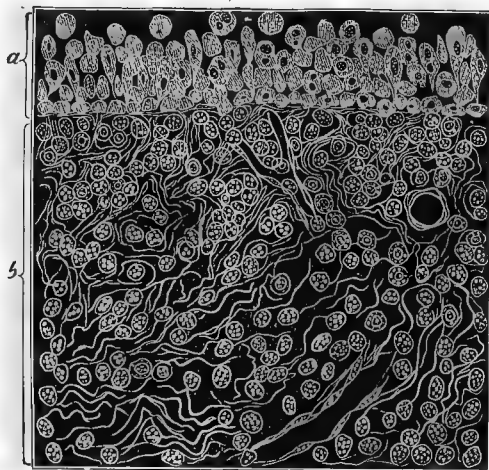


FIG. 79.—Bronchus (medium sized) in acute bronchitis.—(American ox slaughtered at Liverpool.)

- (a) Deep layer of epithelium, germinating and throwing off catarrhal cells.
- (b) Inner fibrous coat, infiltrated with inflammatory cells. (480 diam.)

The columnar epithelium shed.

<sup>1</sup> The basement membrane is not so apparent in the lower animals as in man.

hours after the primary distension of the vessels, and consists in the loosening and desquamation of the columnar epithelium at the foci of greatest congestion.

"The columnar epithelium is thus shed at a very early stage of the attack, and takes *no part whatever* in the after changes which ensue. It is never seen again until the other signs of acute inflammation, such as the distension of the vessels, and œdema of the basement membrane, have passed off. Subsequently we shall see that it is gradually reproduced. The cause of this desquamation of the columnar epithelium seems to be the œdema of the basement membrane loosening its underlying attachments, very much in the same way as the vesicles which form in an acute inflammatory affection of the skin loosen the attachments of the superficial layer of epidermis. The removal of this protective covering from the mucous membrane naturally leaves the latter in an exposed condition, and no doubt the feeling of rawness experienced in acute catarrh of the bronchi is due to the cold air acting upon an over-stimulated and exposed mucous membrane. And, further, it can easily be understood that, where this desquamation takes place to an inordinately great extent, the loss of the ciliary action of the columnar cells will seriously interfere with expectoration, and tend to cause the catarrhal products to gravitate downwards towards the smaller bronchi and air vesicles. This description essentially coincides with what Socoloff found experimentally in animals (Virchow's *Archiv*, vol. lxviii, p. 611), in which he induced an artificial bronchitis by the injection of irritants, such as potassic bichromate, into the air passages. He states that one of the first changes which ensued was the desquamation of the columnar cells, and that they took no part in the catarrhal inflammatory process." This early shedding of the columnar cells, and their non-reproduction until after the subsidence of the inflammatory process, is a fact of real importance, as it goes a long way to explain the occurrence of those caseous tumours mistaken for tubercle, and so often confounded with that growth.

The pneumonic process, which may supervene either by extension of the inflammatory process from the tubes to the alveoli, or the irritation of inhaled inflammatory products subsequent to collapse, is, in the earlier stage, commonly limited to scattered groups of air vesicles, hence the term *lobular* which is applied

to it. It causes the portions affected to appear as scattered, ill-defined nodules of consolidation, irregular in size, and passing insensibly into the surrounding tissue, which is variously altered by collapse, emphysema, and congestion. These nodules are of a reddish-grey colour, faintly granular or smooth, slightly elevated, and soft in consistence. As they increase in size they may become confluent; and in a more advanced stage they become paler, drier, firmer, and to some extent resemble ordinary grey hepatization.

Microscopically examined, they are seen to consist of cellular elements accumulated in the alveoli.

The disease may, as already remarked, terminate fatally by the absorption of the putrescent catarrhal products, by gangrene of the collapsed lungs, or by sudden effusion of fluid into the bronchi, constituting what is termed suffocative catarrh. If a fatal termination does not ensue, the contents of the alveoli undergo degeneration, and are gradually removed, by discharge, or by absorption; or, by coalescence, form caseous masses, which may become encapsuled, undergo the calcareous change, and thus become innocuous; or may induce a diathesis favourable to the actual development of tubercle in the ox, and to symptoms

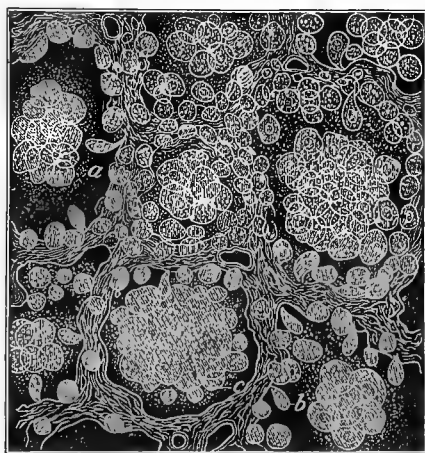


FIG. 80.—Acute catarrhal pneumonia (American ox).—Section through several air vesicles. Shows the alveolar cavities filled with large granular catarrhal cells (c). (b) Catarrhal cells sprouting from the alveolar wall. (a) Coagulated mucus in which the catarrhal cells lie.—(480 diam.)

simulating phthisis pulmonalis in the horse—that is to say, an



accumulation of catarrhal products, epithelial and other cells, within the pulmonary alveoli; cellular infiltration and thickening of the walls of the alveoli and bronchi; increase in the interlobular connective tissue, with, in some instances, the occurrence of fibrinous masses, intermixed with leucocytes in the alveoli, as demonstrated by Zenker of Dresden, but without the occurrence of tubercular tumours (grapes) in the serous membranes and parenchyma of organs.

In all cases of bronchitis the bronchial glands undergo some change. In the earlier stages they are increased in size, contain the products of the bronchitis conveyed by the lymph tract; become more or less friable in consistence; and in more advanced bronchial inflammation distended with catarrhal elements; both glands and contents undergoing the caseous metamorphosis, the products of which may either liquefy or become infiltrated with calcareous matter.

#### TREATMENT.

Venesection is to be avoided; indeed bronchitis is characterised by depression and debility from its earliest stages. In the very commencement of the disease the irritation is generally modified by a moderate dose of opium. In the horse, ox, and sheep remedies termed expectorants are quite useless, having no effect. In the dog, however, they are said to act. They are antimonial wine, ipecacuanha, hyoscyamus, &c.; but I do not think that they are of much service, and are not to be compared to chloral hydrate in from two to four grain doses. The great principle of treatment, however, in all animals after the very earliest stage of the disease has passed off—when, and then only, it may be possible to cut short the attack by a full dose of opium—is to assist in promoting the natural course and termination of the inflammation. It has already been pointed out that in the earlier stages the bronchi are dry, and that subsequently they become moist; and as the moist stage becomes, as it were, matured, the irritation disappears. Such, then, is the natural course, and the practitioner is to assist in promoting this by causing the animal to inhale steam, medicated or simple, the medicated steam being made by adding camphor, creosote, or carbolic acid to the hot water. When the discharge is profuse,

indicating the involvement of a large area of tubes, and a condition of real danger, the inhalation of steam is of the utmost importance, as such inhalation modifies the viscosity of the catarrhal fluid, and facilitates its discharge from the bronchi. And in order to prevent as much as possible the putrefaction of the catarrhal product, such steam should be medicated with carbolic acid. In addition to this the sides are to be bathed with hot water, and rubbed over with oil or a weak liniment to keep off the sensation of cold. If the bowels are costive, enemas are to be administered, but on no account are aloetic purgatives to be given; for obstinate constipation—a very rare complication—a moderate dose of oil may be given. It is far better, however, to keep the alimentary canal in proper order by enticing the animal to eat laxative food, such as linseed mashes, carrots, or grass, if in season. Some practitioners recommend that sulphate of magnesia be given in four-ounce doses daily in the horse's water until the bowels respond. For my own part I have found that horses generally refuse to drink such water, although they may suffer much from thirst; and I generally content myself with ordering half-ounce doses of nitrate of potash twice daily in the water or mash. Good nursing, warm clothing, pure air, and a good stable or loose box, are essentials which are not to be forgotten.

In the course of bronchitis, it will frequently be observed that symptoms simulating those of diabetes insipidus become developed; the animal becomes very thirsty and urinates profusely. These need not cause any alarm; they are due to the absorption and elimination of effete materials and various salts—chloride of sodium particularly—which have been retained, probably in the inflamed part, during the active stage of the disease. If the patient be freely supplied with water, and enticed to partake of good food, the diabetic symptoms pass off, and the animal will speedily become convalescent.

Should debility remain, with cough and irritation, hyoscyamus and tonics are to be administered; and if the case threatens to become chronic, a blister may be applied to the throat and breast, and the iodide of potassium prescribed.

Chronic bronchitis in the horse may cause what is termed thick wind, chronic cough, and eventually emphysema from rupture of the rigid and altered air vesicles and small bronchi.

Metastatic or embolic broncho-pneumonia results as a sequel to septic metritis, absorption of products from septic wounds, or from impure drinking water or tainted food.

#### CHRONIC BRONCHITIS,

As indicated by a loud, metallic cough, emaciation and debility, is not an infrequent disease, particularly in horned cattle, and is due to thickening of the bronchial and alveolar walls, and to caseous masses. It is best treated by hydrocyanic acid, in doses varying from ℥ xx. to ℥ lx., Scheele's strength, in combination with nitrate of potash and bicarbonate of soda, twice per day, a strong blister to the breast, and careful housing and nursing. A morbid change, termed bronchiectasis or dilatation of the bronchial tubes, arising from a chronic interstitial pneumonia, induced by acute croupous pneumonia, broncho-pneumonia, pleurisy, and the inhalation of solid irritating particles, is witnessed in the human being. It is characterised by thickening of the interlobular septa and alveolar walls; and when the fibrosis is extensive, the lung is diminished in size, the tissue is smooth, dense, being in parts almost cartilaginous in consistence, and irregularly mottled with black pigment. The alveolar structure of the lung is in most parts destroyed, and on section the dilated bronchi are seen as numerous large openings scattered over its surface. The dilated bronchi frequently become the seat of secondary inflammatory processes, which may lead to ulceration, and ultimately to extensive excavations of the indurated tissue; but there is a complete absence of caseous changes. The pleura is almost invariably thickened and adherent, the new formation in the earlier stages usually contains new blood-vessels, but later the tissue contracts, and the vessels become destroyed; the contraction of the false membrane may also induce deformity of the chest, and twist the neck to one side. I have seen a case similar to the above description in a dog in which the lungs contained quantities of sand. This dog had been several years in Africa hunting with its master. Before death it was observed that the right side of the chest was depressed, and this was found to be due to carnification and contraction of the adherent lung and connecting new formation.

## CHAPTER LX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (I.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

#### CHRONIC EMPHYSEMA OF THE LUNGS.

CHRONIC EMPHYSEMA of the lungs is of two kinds, namely—(1.) *Vesicular*, due to enlargement and dilatation of the air cells, with gradual effacement of the blood-vessels distributed over their walls. There is anæmia of the affected parts, a tendency to dilatation of the right cardiac ventricle, and a disposition to anasarca of the limbs. The dilated cells vary in size, and when very large it is probable that several vesicles are dilated into one cavity by rupture of the vesicular walls and partitions. These dilatations may be clearly seen through the pulmonary pleura, as they protrude from the surface of the lungs; they are pale, sometimes quite white, the tissue drier than natural. Vesicular emphysema is seen in chronic broken wind, and is due, as already explained, to degeneration of the bronchial tissues. It is, however, a result of bronchitis, and several theories are put forward to explain the origin of emphysema. Dr. Elliotson considers that a want of due expansion of the lungs is the most common cause. He says—"Whatever prevents any one part of the lungs from expanding when the thorax expands, whether it be a material obstruction of the bronchial ramifications or a compression of them, or whatever else, it will occasion those parts which remain dilatable to keep dilated in a corresponding increased degree, in order to fill up the vacuum which the expansion of the chest occasions. When we inspire we dilate the chest, and the air rushes down the trachea, and the lungs follow

the dilated portions. If there be any part of the lung that will not dilate, then other parts are over-dilated to fill up the vacuum, and in that way those parts which we distend are *over-distended*, in order to compensate for the want of distension in other parts; and when once *over-distended* they are often unable to recover themselves, just as is the case in other parts of the body—the urinary bladder for example.

2. *Interlobular Emphysema*, or accumulation of air or gases in the meshes of the connective lung tissue, may arise from rupture of the walls of the air vesicles. This result of rupture is, however, an uncommon occurrence, for generally, when the cells are ruptured, they break one into another, and form permanent enlargements, with rigid walls, by coalescence; but acute emphysema is, however, seen in various diseases, especially of horned cattle, from rupture of air cells or from the evolution of gases, which infiltrate and distend the pulmonary areolar tissue. It may be induced artificially by the injection of the bicarbonate of soda into the veins. It is also seen in an animal which has been destroyed by “blowing,” *i.e.*, by forcing air into the jugulars.

Snarry, of York, in a private letter to me, records several cases of subcutaneous emphysema in cattle, the symptoms of which were as follows:—

The breathing is short, catchy, the air expelled by a double effort, and often associated with a grunting or moaning expiratory sound. In most cases the head is drooping, eyes dull, and the ears flaccid; whilst in others the animal manifests great anxiety, and even some amount of dread or terror, as if each respiratory movement were a source of pain, and possibly there is actual pain, caused by the tissues being dissected by each ingress of air; indeed it may be said that when the patient has completed one respiratory movement it seems to dread the pain of the next. There is always some stiffness of gait, particularly when turned round, and in many instances a subcutaneous emphysematous swelling makes its appearance, sometimes on the first day, but more frequently on the second, third, or fourth day, or even later, after the manifestation of illness. This emphysematous swelling may be first noticed on one or both sides of the chest, the loins—one or both loins; but it gradually, sometimes quickly, extends from its original seat to neck, head,

back, sides,—in fact all over the body. The crepitations may not be very pronounced at first, probably from the fact that the emphysema is deep-seated, and mechanically pushing the superimposed tissue in the outward direction; but in a very short time the crackling sounds emitted on pressing the parts leave no doubt as to its true nature. The bowels are generally constipated, and some recover when free action of the bowels has been established. Some of Mr. Snarry's cases occurred prior, others subsequent, to parturition. In none of the cases was there any great elevation of temperature, 103° F. being the highest.

#### TREATMENT.

No permanent benefit can be expected from any treatment. Much relief may, however, be afforded by careful dieting, keeping the bowels regular, and when the paroxysms of dyspnoea are very great, by the administration of sulphuric ether, chloral hydrate, or the bromide of ammonium. A horse thick in the wind, or suffering from chronic cough, is an unsound animal.

## CHAPTER LXI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (I.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

#### CONGESTION OF THE LUNGS—PULMONARY APOPLEXY—MECHANICAL ENGORGEMENT.

APART from inflammation of the lungs, which will be described immediately, the lower animals, particularly the horse, are apt to suffer from congestion of the true pulmonary blood-vessels, *i.e.*, the branches of the pulmonary artery. This congestive condition is not only seen during the progress of many diseases, such as laminitis, traumatic arthritis, enteritis, pneumonia, heart affections, and various prostrating epizootics ; but often originates in the horse during severe exertion, more especially in the hunting-field when the animal is not “in condition,” or is taxed beyond its strength. As a question of jurisprudence, the occurrence and results of congestive pneumonia, or, more correctly, pulmonary apoplexy, is a matter of some importance, for many instances have occurred, and will doubtless again occur, of horses dying from this affection within a few days after purchase, and the seller has been more than once mulcted in damages to the full value of the animal.

In order to bring a horse into “condition” for severe exertion, it is necessary that it be gradually trained. First of all, it is for a time walked for one or two hours daily, in order that the locomotor muscles be gradually brought into tone for stronger and severer work ; then it is trotted, and afterwards galloped and sweated. Useless fat is thus removed, and the muscles of locomotion, as well as those of respiration, are brought into a condition and tone which enable them to perform the severest and fastest exertion ; the tone and power of the heart are also

increased and elevated, whereby it is enabled to pump an increased supply of blood to all parts of the body, in order to maintain their strength and integrity during the time they are so greatly taxed. In fact, it may be said that the horse's respiratory organs—wind—circulation, and muscular action, are elevated into a high state of functional perfection by careful training, and a horse in this condition will perform during a long run with the hounds or on the race-course, without danger to his health and life. But if a horse that is not thus trained—not in condition, no matter how good its health might be—is suddenly put to severe and prolonged exertion, when neither its muscular, pulmonary, nor circulatory systems are fit to undergo the fatigue consequent thereon, it will be seen that, first of all, the breathing becomes frequent and distressed; the heart beats tumultuously, but with little impulsive force; the voluntary muscles, consequent upon want of tone and exhaustion, obey the will imperfectly. It goes “all abroad,” as the horseman says. The breathing becomes more and more distressed, and at last it falls, and perhaps dies from actual suffocation, consequent upon the pulmonary vessels being overloaded with non-oxidized blood; and after death the lungs are found gorged with blood, black in colour, and prone to rapid decomposition, giving origin to the expression “black as your hat, and rotten as a pear,” and to the idea that the animal had suffered from some chronic disease. In giving an opinion on a case of this kind, the veterinarian must bear in mind that the blackness, tendency to putrescence, or even deliquescent condition of the lung tissue, are results of acuteness of attack, and not of any previous disease; and it may always be accepted that mere engorgements and blackness, without the formation of an exudate, are positive evidence that the disease is not of long standing.

Another cause of congestion of the lungs is actual want of air in badly ventilated stables, the congestion here arising from the stop-cock action of the pulmonary capillaries already described—(see *Death by Suffocation*)—which contract, and prevent, as it were, as much as possible the passage of impure blood into the left side of the heart and systemic circulation. Pulmonary apoplexy is also termed hæmorrhagic infarction, and, as explained by Dr. Yeo, is “universally admitted to depend on a local impediment to the circulation, such as an embolus impacted



in an artery. There being no arterial anastomosis in the lung, such a plug has a very marked effect. The embolus cuts off the normal supply of blood from the part, and the pressure in the arterial branches beyond the stoppage falls to zero. The blood, however, can still find its way through the capillaries into the branches at the distal side of the plug. The branches of the occluded artery are thus reduced to the condition of occluded veins, and as they have none but capillary connections, they may be said to form blind ends to the adjacent arteries. The blood then trickles into these arterial branches and fills them, but no outward flow can take place, therefore they become intensely engorged with stagnant blood. Under these circumstances, the inner coat of the vessel is deprived of its nutrition, for which the constant renewal of the blood is required. This starvation of the minute vessels renders them unfit for their function; they lose their power of retaining the blood, which escapes into the neighbouring textures, forming the dense black consolidation now known as hæmorrhagic impaction."

#### SYMPTOMS.

The symptoms of pulmonary apoplexy are very distressing. The animal stands with outstretched legs, and seems to fight for breath; the nostrils will be seen opening and closing quickly, the flanks to heave rapidly; the eyes are blood-shot and wild, or sunken and dull; there is a tremor all over the body; the legs and ears are deathly cold, and cold sweats bathe the body: the pulse is small and indistinct ("the oppressed pulse of pneumonia" of the old writers), perhaps beating from 100 to 150 per minute in extreme cases; the heart's action tumultuous, but without strength.

In some cases there will be some discharge of frothy blood from the nose; in others hæmoptysis is absent, and if the jugular or other vein is opened, the blood trickles from it, black, thick, and scanty. Death occurs by apnoea.<sup>1</sup>

<sup>1</sup> The term apnoea is used by physiologists to indicate a condition of breathlessness induced by excess of oxygen (*Handbook for the Physiological Laboratory*, by Sanderson and others, page 318), and by pathologists that of suffocation or asphyxia.

## TREATMENT.

In the first place, it is necessary to have the horse so placed that it can obtain as much pure air as possible. It is therefore to be tied with its head to the box or stable door, the body is to be smartly rubbed, not knocked, with wisps of hay or straw, and afterwards warmly clothed. When the surface of the body and extremities are very cold, I have found it to be a good practice to place the feet in hot water and bathe the legs for several minutes, then rub them over with a stimulating embrocation, and bandage them in thick flannel as high as possible. If proper bandages cannot be obtained, hay or straw ropes are to be substituted. The medical treatment must be that which is calculated to equalise and balance the circulation, and for these purposes stimulants are to be recommended, such as the ethers, spirits, wine, or even ale when nothing else can be obtained. I have also found that the tincture of arnica, in one or two ounce doses, acts very satisfactorily; it seems to stimulate the cutaneous circulation, and on this account is well calculated to restore the equilibrium of the circulation. If the congestion does not give way to the above treatment, bleeding is to be resorted to, in order to relieve the pressure on the pulmonary vessels, and the engorgement of the great veins and right side of the heart; from four to six quarts of blood may be withdrawn with safety, but it is better not to repeat it. I am of opinion that it is a cruel practice, and one calculated to do harm, to apply mustard or other powerful irritant to the sides. Horses, when thus treated, begin to be excited in a few minutes after the irritant has been applied, paw, move about, lie down, and perhaps roll in pain; the breathing becomes more accelerated, and loss of strength rapidly follows. I hold it to be imperative that the veterinarian should enjoin perfect quietude and repose, and if anything is to be applied to the sides or breast, let it be warm water, in order to soothe and calm the distressed sufferer.

After the symptoms of congestion have passed away, it is not improbable but that they may be succeeded by those of inflammation of the lungs. Bearing in mind the probability of this sequel, the treatment of the animal for some days must be of the most careful description; the diet must be light, sparing, and easy of digestion; the water must be supplied abundantly,

and even in the earliest stage, if the animal be thirsty, water is to be freely allowed; doses of nitrate of potash given in the water or mash; the air of the stable must be abundant and pure—but the horse is not to be exposed to draughts—and the body kept warm by clothing.

#### POST MORTEM APPEARANCES.

The *post mortem* appearances are, congestion of the pulmonary vessels, with rupture of some of them, and extravasation of blood into the parenchyma, constituting pulmonary apoplexy, whilst others are plugged by emboli. The lungs resemble the spleen, and the term splenification has been applied to this condition. When cut into, the lungs present a deep, dark, purple colour, the vessels are filled with dark blood of a tarry consistence, whilst here and there, interspersed throughout the lung substance, darker points are seen, indicating where extravasation has occurred. Although much heavier and more condensed than natural, the lung tissue will generally float in water; thus differing from the condition of hepatization, which is present when death has occurred from pneumonia. The right side of the heart and great veins are filled with blood of a dark appearance and tarry consistence. The left side of the heart also contains some quantity of blood of the same appearance and consistence.

It may be stated that hypostatic congestion may occur *post mortem* or during the death struggle. This always occurs in the most depending part of the lungs, is to be distinguished by its situation and the antecedent symptoms, the animals having died from other diseases, and is not to be confounded with any diseased condition of the lungs.

## CHAPTER LXII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (I.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

##### INFLAMMATION OF THE LUNGS—LOBAR (SPORADIC) PNEUMONIA.

*Synonyms and Varieties.*—Peripneumonia; peripneumonia vera (as opposed to peripneumonia notha, or capillary bronchitis); febris pneumonica; acute pneumonia; chronic pneumonia; lobar pneumonia; interlobular pneumonia (an affection of the interlobular tissue); primary pneumonia, secondary pneumonia (signifying differences in origin); specific or glanderous pneumonia. I retain the terms sporadic and lobar, as being indicative of the origin as well as of the seat of the disease.

##### PATHOLOGY.

Congestion or engorgement of the lungs has been already described as a result of arrested pulmonary circulation arising from over-exertion, debility of the heart's action, embolism, or to any other circumstance preventing the due arterialization of the venous blood, and causing hæmorrhage from or stasis in the terminal branches of the pulmonary artery.

Inflammation of the lungs is a disease in which all the textures of the pulmonary substances are more or less involved; and this distinction may be made between congestive and inflammatory pneumonia, that, in the first the lesion lies primarily in the pulmonary vessels, and, in the second, in tissues supplied by the branches of the nutrient vessels of the lungs, namely, the bronchial arteries, the pulmonary branches becoming subsequently involved, and having no inconsiderable share in

furnishing the exudative materials, which characterise the condition of hepatization or consolidation that occurs in pneumonia.

The various changes which occur in the course of pneumonia are—*1st.* Arterial injection, characterised by a brighter colour, and dryness of the pulmonary tissue, a condition indicated by harsh respiration and crepitating sounds. *2d.* The arterial injection is succeeded by a condition of engorgement, congestion of the pulmonary vessels, and incipient cedema of the lung. The substance of the lung is engorged with blood and bloody serum; externally it is of a dark red colour, and crepitates but slightly under pressure; it also pits under the finger, and is more easily torn than healthy lung; is heavier than natural, inelastic; its cells are filled with liquid, mixed with air. On being cut a large quantity of bloody serosity escapes from the cut surface, which is frothy in the earlier stages from admixture with air. This, along with the commencing consolidation, proves that the effusion has been poured into the bronchial tubes and air vesicles as well as into the pulmonary tissue. During this stage the lung tissue will still float in water, although it is heavier and less crepitant than natural.

If a very thin section be made, and examined microscopically, the capillaries will be found filled with blood; the air vesicles enlarged and granular, with incipient division of their nuclei; and exudation corpuscles, mingled with red globules, which have escaped from the vessels, are seen in the alveoli.

*3d. Red Hepatization.*—If the inflammation continue, other changes occur in the lung substance. The lung is red externally and internally; is solid, sinks in water, and no longer crepitates under pressure; it, however, tears easily and breaks down under pressure; and on this account Andral objected to the term hepatization, and termed the second stage of pneumonia red softening—*ramollissement rouge*. The colour is less livid than in the earlier stage, being a dull reddish-brown, becoming brighter on exposure to the air. This reddish-brown colour is never very uniform, and in some animals diversity of colour is very marked: thus in the ox the inflamed lungs present such a variety of colour as to cause the term “marbled” to be applied to it. This is due to the very distinctly lobulated anatomical character of the ox’s lungs, and the consecutive nature of the lobar pneumonia. Dark red spots indicate the

earlier or second stage and the grey spots the third or later stage of the disease, which is called—

4th. *Grey Hepatization*, termed by Andral *ramollissement gris*, or grey softening. In the horse, perhaps the term grey softening may not be inappropriate, but in the ox solidification expresses the condition much more accurately. The cut surface of the part which has run on to this stage presents a grey tint, the redness of the preceding stage having passed away, and the granular character is less distinct. In the horse, dog, &c. the tissue has very often lost its firmness, and has become soft and pulpy, and an abundant dirty-looking puriform material oozes from the cut surfaces. In the ox the grey portions are generally smooth, glistening, and firm. In some instances the smooth, glistening appearance is observed in the inflamed lungs of horses, but the firmness is rarely so great as in the lungs of oxen, in which the tissues of consolidated parts retain their marbled appearance, and acquire a resisting character, or are transformed into a more or less firm cheesy material. In some instances, however, the grey exudate, even in the ox, is broken down into a more or less pulpy material.

If a portion of hepatized lung be torn, and the torn surface examined with a magnifying glass, the tissue will appear to be composed of a crowd of small red granulations, lying close to each other, air vesicles clogged up, thickened, and made red by the inflammation. In the catarrhal form of pneumonia commonly seen in the horse, the exudative products are mostly accumulated in the interior of the air vesicles, but in the exudative form (croupous) seen in the ox, and in cases of pleuropneumonia in the horse, the exuded materials not only fill the air vesicles but the interstices of the pulmonary connective tissue.

Suppuration of the lung is a more advanced state of grey hepatization; the lung is softer, yellower, and more pulpy, but the condition is not materially distinct from the others, as pus cells are present in the advanced stages of pneumonia, and the distinction between softening, grey hepatization, and suppuration is more one of terms than of reality. Diffuse suppuration of the lungs is not rarely witnessed in newly calved cows; exposed to wet and cold, it runs a rapid course, terminating fatally in three or four days, the lungs being engorged with puriform materials. It is a

remarkable thing that suppuration of the lungs very rarely runs on to the formation of abscesses when the inflammation is not due to a specific cause, such as glanders or pyæmia. I have certainly seen abscesses in the lungs of both oxen and horses, but the event is a very rare one, and Sir Thomas Watson endeavours to account for the rarity of pulmonary abscesses in a very ingenious manner. He says—"When I was speaking of inflammation in general, I pointed out to you the remarkable influence which the presence of air in contact with the inflamed part has in accelerating or determining the event of suppuration. In a recent cut through the skin the admission or the exclusion of the air to the cut surface will make all the difference between the adhesive and the suppurative inflammation; and so in other cases which I then mentioned, and will not now trouble you by repeating. Now it seems to me that the same principle obtains in inflammation of the lung. First, there is an effusion of serum and blood, then of lymph and blood; but the air, passing into the surrounding sounder tissue, and penetrating for a time even the inflamed portion itself, causes the suppurative process to supersede the adhesive; and so no wall of circumvallation is formed by the coagulable lymph, as is the case in areolar tissue when not accessible by the air."—(WATSON'S *Lectures*, page 81.)

Gangrene is more generally a result of congestion than of inflammation of the lungs, but its occasional occurrence in pneumonia is indisputable. Sometimes it occupies a large portion of the lung, and is not circumscribed, sometimes it is more limited. The affected parts are dark, dirty olive or greenish-brown in colour; foetid in odour; moist, wet, and diffuent in consistence. The occurrence of mortification has been ascribed to thrombosis occurring in the branches of the pulmonary artery—(Huss, Carswell)—and to the destructive effect of the inflammatory process destroying the vitality of the tissue, or to an arrest of the circulation by the excessive accumulation of its products in the interior of the air cells.

Pneumonia may be double (bilateral) or single (unilateral), that is to say, it may affect one or both lungs; double pneumonia is, however, very uncommon. I have for many years carefully noted the site of pneumonia, and have found that the inflammation is much more commonly situated in the right than in the left lung, both in horses and in cattle, in epizootic, sporadic,

and contagious pneumonia; that, generally speaking, the inflammation commences in the inferior portions of the lungs; that it invades the tissue from below, upwards; that in all cases the bronchi are more or less involved in the inflammatory process; and that when the pneumonia is superficial, the pleura participates in the inflammatory process, losing its normal colour and translucence, becoming opaque, and covered with a layer of fibrinous material; that the exudation, whether it be into the parenchyma or on the surface, is fibrinous. When the disease originates deeply within the lung substance, the pleurisy may be and occasionally is absent.

When the pulmonary inflammation terminates in resolution, or a gradual return of the lung to its normal condition—and this termination is the most frequent one in ordinary pneumonia, provided the animal be properly treated—the exudates liquefy, undergo fatty degeneration and disintegration, and become so altered that they can be removed by absorption, and as the softened matters become absorbed, the circulation is gradually restored, and the lung slowly attains its normal character.

In glanderous pneumonia, the exuded materials are transformed into an ichorous, infecting, puriform fluid, and in pleuropneumonia-bovina contagiosa they undergo a caseous necrotic change; the inflammatory process meanwhile continuing in other portions of the lungs until a large part of them becomes consolidated, impervious to air, and the animal dies from suffocation and anæmia.

The various stages of pneumonia are manifested by certain sounds detectable by the ear when applied to the sides of the chest. In the first stage, and previous to the occurrence of much engorgement, a crackling sound is heard, mingling with the vesicular murmur, over the inflamed part; the sound is of the smallest and finest kind of crepitation, similar to that emitted by salt when thrown on hot coals. As the inflammation advances the crepitations become more and more pronounced, until they entirely supersede the vesicular murmur. These crepitations do not remain long; they are superseded either by a return of the vesicular murmur, indicating the resolution of the inflammation by a tubular sound, or an entire absence of sound.—(See *Auscultation*.)



## ETIOLOGY.

The causes of sporadic pneumonia are similar to those of bronchitis, laryngitis, &c., namely, exposure to cold and wet, sudden chills, and housing in very cold, draughty stables. Horses kept in ill-ventilated stables are undoubtedly rendered susceptible to many diseases, and to pneumonia amongst the rest, but they will bear impure air even better than cold draughts blowing directly upon them. I have repeatedly observed that the slightest cold contracted by a horse kept in a draughty stable has almost invariably been succeeded by pneumonia, and that if the animal were not removed to a more comfortable situation the disease tended to a fatal termination. It has already been stated that inflammation may succeed the congestive condition induced by severe exertion; it may also be induced by irritating gases; smoke of burning hay and straw; foreign bodies entering the lungs from the bronchi, examples of which are not uncommon in cows, as sequelæ to parturient apoplexy, where medicine finds its way into the trachea and bronchial tubes, and in horses choked by irritating and powerful remedies, such as ammonia.

Direct injuries to the lung through wounds in the thoracic walls are not always succeeded by much inflammation, the lungs appearing to have remarkable powers of recovery from direct injury, provided it be not crushed, or that foreign bodies or extravasated blood are not forced into the wound in its tissue.

Pneumonia may also be induced by any material altering the composition of the blood, epizootic influences, purpura, anthrax; accidental products accumulating in the blood; mechanical or solid materials (thrombi) formed elsewhere, and conveyed to the lungs by the blood, as in phlebitis; infecting influences of materials conveyed to the lungs, as in pyæmia or glanders. It may be the result of passive congestion arising from disease of the heart or weakness of the circulation, induced by exhausting diseases or old age; or it may be the localisation of a specific disease, as in pleuro-pneumonia contagiosa.

The above-mentioned causes must be considered as predisposing causes only. A cold intermittent draught tends to the lowering of an animal's vitality, equally as much as placing a fowl in an ice-box will render it susceptible to anthrax. (It

must not be concluded, however, that ventilation is to be interfered with in any way—*vide* Treatment.) It is extremely probable that all congestions of the lungs are the result of microbic infection acting upon an organism already weakened by unhygienic conditions, over-exertion, improper food, &c., with the sole exception of acute congestion, which is the result of over-exertion; the disease known now as infectious pneumonia is undoubtedly, as its name implies, of microbic origin, but the specific organism is not definitely known. One of the *Pasteurellas* has been considered as the cause, but this is not even certain, and much work is being done on the subject.

#### SYMPTOMS.

The symptoms of pneumonia, namely, the greatly accelerated respiratory movements, oppressed pulse, and other signs of excitement described by Youatt, Gamgee, and others, are not by any means diagnostic of pulmonary inflammation. In many cases the breathing in the earlier stages is not difficult, and one is often surprised upon auscultating the chest to find that consolidation has proceeded to a great extent without any very distinct symptoms having been presented.

In the earlier stages, the symptoms are acceleration of the pulse; in very many instances it will average eighty beats per minute; hotness of the mouth, and elevation of the temperature to 103°, 104°, or even 106°; there is a dry, dull cough, coldness of the extremities, and some degree of rigor. In some instances the disease is ushered in by a severe rigor, succeeded by a hot stage; the mucous membranes are red and injected, the conjunctivæ presenting sometimes a rusty yellow tinge; the animal does not lie down, and if loose wanders occasionally about in a dull, depressed manner, now and then eating a mouthful of food. It is stated that the animal stands with its limbs outstretched and head protruded. I have, however, failed to observe this, except in very acute and congestive cases, until the disease has made considerable progress. There is some degree of constipation, but the fæces are mixed with flakes of mucus; the bowels are irritable, and do not tolerate purgatives. If complicated with pleurisy, the breathing is more distressed, abdominal, and painful, but in pure pneumonia there is an absence of any very painful symptoms. I have carefully noted the breathing in

many cases of pneumonia, and found that the respiratory movements may not be above twenty per minute for several days after attack—a difference so slight from the normal number, that one is apt to overlook it altogether. The observations of Dr. W. Gairdner, that “the dyspnoea of *pure* pneumonia is a mere *acceleration* of the respiration, without any of the heaving or straining inspiration observed in bronchitis, or in cases when the two diseases are combined,” hold good both in horses and cattle; but congestive paroxysms are not at all infrequent during the progress of the malady, during which the breathing is not only greatly accelerated, but also laborious and distressed. As the disease advances, the respiratory movements become somewhat accelerated, until the period of crisis, when usually the breathing becomes much quickened. As a clinical fact, it may be stated that the fever continues for some days—five, eight, or sometimes longer—and that it then gradually subsides, the pulse falling several beats per minute, the mouth becoming cool, the elevation of the temperature gradually subsiding, the appetite returning; the secretion of urine, which during the febrile period had been scanty, of a high specific gravity, loaded with urea, and deficient in chlorides, is now abundant. Whilst these symptoms of returning health are becoming apparent, it will be found that the respiratory movements are increased in rapidity, and auscultation will reveal the fact that consolidation of the lung increases during the subsidence of the febrile symptoms.

This had led some pathologists to conclude that all pneumonias are specific fevers terminating by an exudation into the lung tissue, in the same manner as variolous fever terminates in an eruption on the skin. I cannot agree with this view, inasmuch as the pneumonia is a concomitant condition from the commencement, and I look upon the exudative process as affording relief to the febrile symptoms, in the same manner as an ordinary swelling gives relief in such diseases as lymphangitis and ordinary inflammation of areolar tissue.

It will be seen that pneumonia is dangerous during two stages: first, during the early fever, which may destroy life by its intensity; and, secondly, during the period of lung hepatization, which may prove fatal by so altering the lung tissue as to produce suffocation.

These, then, are the two periods during which the practitioner

has to exercise his skill and care. In some instances death may occur subsequent to the consolidative stage from rapid deliquescence of the inflammatory products, extensive suppuration, or contamination of the blood by degraded exudates absorbed into the circulation.

In human medicine, the absence of the chlorides in the urine during the earlier stages, and their return in increased quantities in the convalescent stage, is looked upon as of great importance. This is a matter which requires further observations in veterinary practice. In most instances, a discharge of a rusty-tinged more or less viscid material from the nose is observed in the horse. This is sometimes streaked with blood; it is not, however, a constant symptom, as in all probability much of what might be expectorated by the human being is swallowed by the lower animals. The character of the discharge is significant; in bronchitis and catarrhal pneumonia it is more or less purulent, and if excessive, indicates a condition of great danger. In croupous pneumonia it is amber coloured, viscid, but not abundant.

It is agreed upon by all observers that a horse will not lie down during the intensity of any chest disease. This is not a diagnostic sign of any particular affection of the horse, as it will persistently stand while suffering from many other maladies.

The ox will lie on the sternum during all the stages of pulmonary diseases.

When the breathing is very rapid, the horse will stand with the elbows turned outwards, and the toes turned inwards. If great prostration succeeds, the position of the limbs is altered. It will then stretch its feet apart; the elbows will be turned inwards, and it will balance the trunk upon the extremities.

The pulse of pneumonia is very variable; sometimes full, with a degree of hardness; sometimes full and soft; at other times it is small, irregular, intermittent, or double—the latter condition marking a congestive condition of the pulmonary vessels and right side of the heart and veins, as well as an anæmic state of the arterial system.

In ordinary pneumonia, running its course to a favourable termination, the period of consolidation, which is indicated by absence of sound, or a small degree of tubular breathing, is succeeded by the advent of secondary and larger crepitations, which increase

in number and intensity for two or three days, then, gradually diminishing, are superseded by the normal respiratory sounds.

To sum up, it may be stated that the diagnostic signs are those revealed by auscultation and percussion. First of all, small crepitations, indicating injection of the nutrient arteries and dryness of the lung tissue; secondly, tubular breathing, or absence of sound, indicative of engorgement and consolidation; and thirdly, the reappearance of the crepitations, now of a larger character, pointing out that the exudate is undergoing metamorphosis, and becoming absorbed.

The secondary crepitations are sometimes of a bubbling character, and if associated with sunken eyes, a wrinkled expression, and feebleness of the pulse, indicate a condition of great gravity, either of extensive suppuration or of moist gangrene. Both these conditions may be associated with fœtor of the breath, the latter particularly being accompanied by a horrible odour.

During all the stages of pneumonia, increased dulness is elicited by percussion.

Inflammation of both lungs, or double pneumonia, must be regarded as a source of very serious danger from the great impediment it presents to the breathing, and deaths from it are much more numerous than from the single form. It is, however, very rarely met with.

Pneumonia is sometimes latent, and may remain undiscovered perhaps until after the animal's death. In all cases of ill-defined maladies, it is necessary that the chest be thoroughly examined, when disease of a very grave nature may occasionally be detected, although the animal has never presented any very evident signs of serious lung disease.

During the prevalence of epizootics, pneumonia is apt to assume a marked adynamic or typhoid form, attended with an early breaking down of the pulmonary tissue. This is, however, rare, and the most common form of chest disease met with is a combination of pneumonia with pleurisy, in which the symptoms of both diseases are inseparably blended.

Acute pneumonia, especially if associated with bronchitis, is sometimes complicated with laminitis or inflammation of the feet.—(See *Principles and Practice of Veterinary Surgery*.) Acute inflammation may terminate in a chronic form of pneumonia, in which the inflammatory products undergo fibrous or

caseous degeneration, keeping up a continued degree of irritation, unfitting the animal for work, and gradually destroying life by inducing anæmia, glanders, or hydrothorax. The symptoms of non-contagious pneumonia and pleuro-pneumonia in the ox are much more acute than those of pleuro-pneumonia contagiosa.

*Hæmoptysis*, or bleeding from the lungs, is not generally associated with pneumonia, but may be witnessed in congestion, accidental rupture of vessels, glanders, purpura, and in a passive form in some epizootic disease or influenza; in which case I have seen the blood of a dark colour, and not frothy as in ordinary hæmoptysis.

#### TREATMENT.

*Bleeding.*—The abstraction of blood was considered the sheet-anchor in pneumonia. Percivall says—"I will take it for granted that pneumony, either in its congestive or inflammatory form, has set in; which being the case, it becomes the imperative duty of the practitioner, without any regard whatever as to the state of the pulse or the condition of his patient, to abstract blood the moment he is called in. Generally speaking, a large orifice in the jugular vein is to be preferred to a small one; in cases of imminent danger it is absolutely indispensable. The quantity of blood to be abstracted must be as great as the patient will bear; and our sure guide in this is the effect which the efflux of blood has upon the pulse at the jaw. While the blood is flowing, keep your fingers applied upon the submaxillary artery; so long as you feel that pulsating, so long may the stream of blood be continued; but the instant the vessel collapses under the pressure of the fingers, and pulsation is no more perceptible, let the blood-can be removed and the vein pinned up. . . . The quantity of blood we shall be able to draw on the first occasion will vary in different subjects and under modified circumstances; it may amount to a couple of gallons, it may not exceed a couple of quarts. . . . A second blood-letting is often borne better than a first. When the quantity taken in the first instance has been but small and inadequate, if we will only wait a few hours until reaction appears to have taken place, we shall commonly be able fully to accomplish our object. Six, twelve, or twenty-four hours after the first full blood-letting, guided by the

exigencies of the case, principally by the state of the breathing and pulse, it may become necessary to repeat the bleeding, regulating the quantity, as before, by the perception of the pulse at the jaw. I have found it requisite to bleed thrice during the first eighteen hours.”—(PERCIVALL’S *Hippopathology*.) This was the belief and doctrine not only of Percivall, Youatt, and others, but of Professor Dick, who taught and maintained its correctness up to the time of his death. A glance at the pathology of the disease, no less than actual statistics, will enable any unprejudiced observer at once to see the fallacy of the doctrine; and veterinary surgeons, with but few exceptions, have been long convinced that bleeding most materially increases the mortality of the disease. The experience of the medical profession has led them also to arrive at a similar opinion, and Dr. Wilson Fox, in Reynolds’ *System of Medicine*, sums up the conclusions upon this head as follows:—

“ (1.) That indiscriminate bleeding immensely increases the mortality of the disease.

“ (2.) That it is especially fatal in old people and young children, in patients of exhausted constitutions, and in those suffering from chronic diseases, and particularly in Bright’s disease.

“ (3.) That it is absolutely unnecessary in the majority of cases of young adults and also young children.

“ (4.) That in the vast majority of cases it has no influence whatever either in cutting short the disease, or in lessening its duration or diminishing the pyrexia, but that occasionally these results appear to follow its use when practised early.

“ (5.) That in the majority of cases it hinders the critical fall of temperature and delays convalescence.

“ (6.) That in the majority of cases, as shown especially by Dr. Bennett’s and Dietl’s data, recovery is equally, if not more rapid, when it is not practised as when it is resorted to.

“ (7.) That in a few cases a moderate venesection may be necessary in the early stages to avert immediate danger of death from asphyxia.”

The above conclusions have reference to moderate bleeding only, repeated bleedings being condemned by Dr. Fox as “a system whose impropriety it is scarcely needful to discuss further.”

Bleeding, then, can only be safely practised in the very earliest stages of a few exceptional cases, in which symptoms of dyspnoea are very urgent; and even then it must not be pushed so as to debilitate, for more horses die from the prostration of strength at later periods than from the occasional suffocative effects of the earlier and congestive stages.

Bleeding in some cases, no matter when it is practised, seems to afford a relief to the breathing, but this effect is only temporary, and disappears in a very short time, the rapidity of the respiratory movements in pneumonia being dependent on oedema or consolidation, conditions upon which the withdrawal of blood can have but little or no effect.

Convinced of the inutility and danger of venesection, many veterinarians, undoubtedly influenced by the teaching of Dr. Tod, fell into the other extreme, and treated pneumonia by large and repeated doses of stimulants. What possible good effect this kind of treatment has upon an ordinary case of pneumonia is beyond my comprehension; it can only add to the irritation of the inflamed part, and increase the amount of exudation, if pursued in the earlier stages. I have seen it extensively tried, and must confess the results have been most disastrous. In the later stages, during the deliquescence and absorption of the exudate, if the pulse be small, or in any degree presenting the double or dicrotonous character, when the system is depressed by the obnoxious effects of large quantities of effete materials in the blood, moderate doses of stimulants are both necessary and beneficial. The practitioner should, however, wait until the consolidative stage has to some extent disappeared, and secondary crepitations established. Four or six ounces of whisky or brandy may then be given two or three times a day; not every hour, as recommended by some practitioners. The effect of the first doses must be carefully noted; if they stimulate the appetite, they are to be repeated; but if they cause distress in the breathing, or any signs of exacerbation, they are to be discontinued.

Calomel, opium, digitalis, tartar emetic, seem to have no effect in curtailing the duration of the malady. Opium is only admissible when the pneumonia is associated with pleuritic pain.

Purgatives are inadmissible in the horse on account of the sympathetic and irritable condition of the alimentary mucous membrane.



If constipation be extreme, enemata, or a small dose of oil, are to be preferred to the more active cathartics. Aloes is contra-indicated, and should never be given. In horned cattle a saline cathartic may be given in the early stages, for the purpose of unloading the stomachs and alimentary canal, and thus give greater freedom to the respiratory movements.

Nitrate of potash given dissolved in the animal's drinking water seems to diminish the amount of exudation, and modify the fibrinous condition of the blood. It has also a cooling or detergent effect upon the system generally, modifying the pyrexia, and, by acting upon the kidneys, assists in the excretion of effete materials from the blood. In some instances, particularly when the kidneys remain torpid, its diuretic effect is increased by a few doses of colchicum. But the administration of the nitre or the colchicum should not be persevered in too long, as they both tend to debilitate, and whenever free diuresis is established, they should be discontinued. In some instances, particularly if the patient be debilitated or out of condition, spirits of nitrous ether may be substituted for the potash salt.

*Blisters.*—For many years I have held that the application of the so-called counter-irritants, whether they be cantharidine blisters, mustard, turpentine, or any other powerful irritant, is not only useless but dangerous in the earlier stages, and uncalled for when convalescence is progressing favourably.

In the earlier stages, they add to the distress, prevent the freedom of the respiratory movements, and increase both the fever and fibrinous condition of the blood; and large cantharides blisters, the cantharidine of which is absorbed into the blood, induce a condition of blood poisoning characterised by albuminuria and great prostration, and by irritating the urinary passages increase both the distress and the fever. I am glad to find that this conclusion—a conclusion, I may state, which has met with much opposition—is endorsed by physicians. Dr. Wilson Fox says—“*Blisters*, in the earlier stages of pneumonia, are to be considered as both useless and as greatly increasing the distress of the patient. When resolution is progressing favourably, they also appear to be quite unnecessary. In a few cases when resolution is delayed, or when there is evidence of a small amount of pleuritic effusion, they may, I believe, in adults be occasionally employed with apparent advantage. In children

they are almost invariably inapplicable. Warm fomentations or poultices to the sides often give great relief to the pain. I have by no means satisfied myself that any advantage accrues during the acuter stages from any more irritant applications, whether mustard or turpentine, though in cases of threatening collapse, or when dyspnoea is severe, they have occasionally appeared to afford relief." — (See REYNOLDS' *System of Medicine*, vol. iii., p. 701.)

In the horse the application of either mustard or turpentine causes very great distress, a high state of excitement, an increase of the febrile disturbance, and often tends to hasten a fatal termination. For these reasons the indiscriminate employment of such remedies is highly condemnable. Fomentations to the sides, however, afford relief; and they should be persistently employed in all cases marked with rapid or distressful breathing.

When the extremities remain very cold, the return of warmth and circulation will be much assisted by frictions, or the application of a mild embrocation and bandages; and, to sum up, it may be stated that the successful treatment of pneumonia consists in the following essentials:—1st. Placing the patient in a well-ventilated loose box, where the air is pure, abundant, but not too cold. Within the last few years, the administration of oxygen by inhalation has found much favour—the respirations, the pulse, and the general condition of the patient being much improved after each inhalation of ten minutes' duration. If the air be impure, suppuration of the lungs to an extreme extent will be apt to be established, as impure air contains organic germs in great abundance, and these, gaining access to the inflamed part, increase the tendency to the formation of pus. In the winter months, especially if the weather be cold, I have noticed that coldness of the air increases the pulmonary inflammation, and that after a very cold night an animal has been in a state of approaching collapse in the morning. To prevent the occurrence of the mortality from the above cause, veterinary establishments ought to be furnished with loose boxes, where the air can be artificially warmed to a temperature of at least 65° F. 2d. The surface of the body, extremities, and head are to be kept warm by suitable, but not too heavy, clothing; frictions, and the application of non-irritating stimu-

lants to the extremities, being useful in cases where they are deathly cold. 3d. The horse is to be allowed as much cold water as it will drink; but it is not advisable to allow it to drink a large quantity at once. If it be permitted free access to water whenever it pleases, by placing it in its box or stall, it will generally partake of it in sufficient but not over-abundant quantity. 4th. The febrile condition must be treated according to the intensity of the symptoms. If the fever be high, nitrate or chlorate of potash is to be administered, but if, on the contrary, the fever be of an adynamic type, carbonate of ammonia with camphor may be given in the form of a ball, in addition to the potash salt, and it is found that hyposulphite of soda in the drinking water is of great value. 5th. If the bowels be constipated, they are to be carefully regulated by a moderate dose of oil, and by enemas. 6th. During resolution of the inflammation care must be taken that nothing be done to check the action of the excretory organs. Should moderate diarrhœa or increased diuresis come on, they are on no account to be checked, as they are merely critical discharges, whereby the effete materials in the blood are eliminated from the economy. 7th. In the earlier stages the food is to be simple, laxative, cooling, and nutritious. Bran and boiled linseed mashes, a moderate allowance of good hay, roots if in winter, or grass if in summer. During the later stages strong food, as oats, beans, with grass or roots, are to be allowed in moderation; and should the appetite be bad, stomachic stimulants, as spirits of nitrous ether, with gentian or nuxvomica, are useful adjuncts; but should the patient refuse to feed, milk and eggs are to be freely supplied. This it will drink if stinted in its water; it should never be forced upon it by horning, nor should gruels, hay tea, large doses of ale, porter, &c., be forced upon it.

I may state, in conclusion, that in all cases where depression and debility are prominent symptoms, with a small, feeble, rapid pulse, stimulants should be cautiously used, and if after a few doses they appear to do no good, they are to be discontinued, and the case left without much meddling or interference; and that bleeding is but rarely called for, and only in cases where the dyspnœa is great, and the danger to life imminent. Even then it is not to be pushed to the extreme recommended by some writers and teachers.

It must be borne in mind, as already pointed out in the chapter on Degenerations, that pneumonia, pleurisy, bronchitis, and other diseases, even of a mild type, are apt to terminate fatally in overfed and pampered animals, such as those kept for show purposes, no matter how discreetly they are treated ; for in such animals the vital organs—heart, liver, kidneys, &c.—will be found in such a state of degeneration as to prevent the performance of their functions under the altered conditions of induced disease. So long as the animals are not subjected to any trivial cause of disease, function seems to be performed ; but whenever the system is subjected to any trial, such as a chill, fatigue, parturition, &c., the powers of the altered organs are wholly or partially lost, and death is the result.

## CHAPTER LXIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (1.) DISEASES OF THE RESPIRATORY ORGANS—*continued.*

##### PLEURISY—PLEURITIS.

INFLAMMATION, partial or general, of the serous membrane that lines the cavity and covers the viscera of the thorax, attended with effusion of serum, exudation of lymph, or, rarely, the formation of pus.

Pleuritis, or a combination of it with pneumonia, is the most common form of chest inflammation met with during the prevalence of the easterly winds of spring and early summer, and has been fully described at page 405.

In the lower animals, exposure to cold is the most common cause of pleurisy. It has followed clipping when an animal has been exposed to cold, and Duvieusart states that he has seen three hundred cases of pleurisy in a flock of sheep shorn in February, thirty of which died.—(GAMGEE'S *Domestic Animals*.) It will sound strange to veterinary surgeons to be told that cold is now believed to have no effect in causing pleurisy in the human being : such, however, is the belief.

Pleuritis in the horse often partakes of a rheumatic character ; the inflammation being induced by the same cause—cold—and assuming a similar metastatic type and character, changing its seat from the pleura, sometimes to the pericardium, endocardium, or to the ligamentous or tendinous structures of the extremities. In more than one instance navicular disease has succeeded an attack of pleurisy, and I have one specimen in my possession showing ossification of the heart

caries and eburnation of the navicular bone, as results of acute navicular arthritis succeeding an attack of rheumatic pleurisy.

At the outset the inflammatory phenomena commence in the subpleural tissue, the blood-vessels of which become congested. At first the congestion presents an irregularly spotted appearance; the spots, however, multiply rapidly, and the red colour becomes diffuse by their confluence.

In other instances the pleural surface presents a streaky appearance, whilst in others the whole of the pleura, pulmonalis and costalis, presents a bright red congested appearance over its surface. The secretion of the pleural surfaces is at first suspended, and if the ear be applied to the side of this stage a dry friction sound will be heard.

The dryness of the membrane is very shortly succeeded by effusion of serum, and the formation of an exudate which has a tendency to become organized into a false membrane, causing the adhesion of the two pleural surfaces.

It is important that the time in which these false membranes may be formed be clearly ascertained. The experiments of Andral throw some light upon this subject, which is important to the veterinarian as a question of jurisprudence.

Andral made experiments upon the pleura of rabbits by injecting acetic acid into them.

He sometimes found at the end of nineteen hours soft, thin, false membranes, traversed by numerous anastomosing red lines.

In other rabbits, placed under circumstances which appear to be exactly similar, no such result had taken place at the end of a much longer period; and the pleura contained only a serous or puriform liquid, mixed with unorganized flakes of lymph.

Experiments performed by myself upon healthy subjects (horses) in 1874 proved that a false membrane, presenting some degree of organization, may be found in twenty-four hours after the injection of an irritant into the pleural sac, and that the puriform appearance mentioned by Andral is an evidence of an inflammation of longer standing, arising from degeneration of the inflammatory products.

It may be briefly stated that all cases of pleuritis terminate either in the exudation of lymph, which may liquefy and be re-absorbed, or in the formation of a permanent false membrane. But the most ordinary result is effusion of serum,

constituting a limited hydrothorax, even to invasion of the walls of the thorax, a natural result of a diffuse and intense inflammation; and that in pleuro-pneumonia contagiosa of the ox there is often cedema of the thoracic walls without actual hydrothorax.

Pleurisy may be either double or single; generally it is single, and confined to the right side.

Pleurisy is sometimes, but very rarely, caused by direct violence to the thoracic walls. Pleurisy is often secondary to other diseases, such as septic broncho-pneumonia—the transit or corn-stalk disease of American cattle—and of many forms of blood poisoning.

It has been said that all pleurisies in the horse are the result of invasion by the streptococcus of strangles, and that a similar condition in man is always the result of tubercle bacilli. It is, perhaps, going a little too far to say this, as although streptococcus may often be the cause of pleurisy in the horse, it is also frequently found that a non-purulent exudate may exist. It is probable that pleurisy, as in the majority of other inflammations, is directly the result of microbic invasion, but no particular organism can be credited with the cause in all cases. The same may be said of rheumatic affections. This is a condition also most probably the result of bacterial infection, and a condition in which the serous membranes are particularly liable to attack, so that occasionally the pleura suffers.

#### SYMPTOMS.

The disease at the outset is characterised by some degree of rigor, very often a mere chill, manifested by a staring coat, and coldness of the surface of the body. This is, however, succeeded by signs of pain, often mistaken for colic, during which the horse paws, and perhaps lies down and rolls; it eventually becomes stiff and sore, and if suddenly approached, or if rapped upon the affected side, will groan. The acts of respiration are performed rapidly and incompletely, the ribs are fixed, the respiration is mostly abdominal, and a hollow line extends along the inferior border of the false ribs, from the sternum across the flank to the anterior spine of the ilium. A

dry, short, painful cough supervenes; the pulse is hard and quick. If the side be closely examined, the muscles covering the affected part will be noticed to tremble or quiver; this lasts but a short time only, and is succeeded by diminished motion. These signs, as well as the pain upon pressure, indicate that the intercostal muscles participate in the inflammation, and that pleurisy is generally associated with pleurodynia, first described by Haycock.

When the pleurodynia, or inflammation of the muscles, is very intense the animal moves in a very rigid manner, steps slowly and very short; is greatly dejected, the back is arched, the skin exhibits great tenderness when subjected to pressure. I may state that I have seen some cases that were so stiff and sore as to fall when compelled to move.

*Hydrothorax*.—When an animal dies of pleurisy, especially if it has survived the first stage, a considerable amount of effusion of serum will be found, constituting dropsy of the chest, or hydrothorax. In some instances a large quantity may be found in a day or two, and in some rare cases without any perceptible disease of the pleura. The symptoms of hydrothorax are short, quick, laboured respiration; the pulse small, quick, soft, often intermitting; auscultation reveals absence of sound in the inferior part of the chest, or a sound resembling that of drops of water falling into a well, as has been already explained. The hydrothorax in the horse generally invades both sides of the cavity of the thorax, for a communication exists owing to the loose diaphanous or web-like structure of the mediastinum, as pointed out by Rigot, Delafond, and Bouley. This natural communication is in some instances obliterated by exudation of lymph on the mediastinal surface, in which the hydrothorax will be confined to the one side. In other instances the serous effusion, confined in sacs of adventitious products, will be confined to certain circumscribed parts of the chest only. The liquid effused is composed of serum mixed with flakes of lymph, and is generally more or less clear upon the surface, turbid in the lower parts from admixture with particles of exudative materials; it is sometimes, however, tinged with blood. If the clear liquid be placed in a glass and left at rest, it generally separates into clot and serum, proving that it contains the constituents of fibrin, which coagulate on exposure to the atmosphere.



In forty-three cases observed by M. St. Cyr, the effusion from the first to the seventh day presented a port wine colour in nine cases, a sero-sanguineous appearance in six, muddy or greyish in three. From the eighth to the fifteenth day the port wine colour was observed in two, the sero-sanguineous in three, muddy greyish in four, and limpid in six. From the sixteenth to the thirtieth day the colour was limpid in five cases, and after the thirtieth it was also limpid in three cases. M. St. Cyr draws the conclusion that the liquid begins to clear up towards the end of the second week, and that it is clear after the twenty-fifth or thirtieth day, and that this is the epoch from the acute to the chronic state of pleurisy.

English veterinarians generally maintain that the advent of hydrothorax is manifested by an apparent improvement of the pleuritic symptoms. They state that the pulse falls, the breathing becomes easier, &c. This is so far true, but is not diagnostic, as the abatement of the symptoms indicates the subsidence of the pain and fever, and that the dry condition of the pleura has passed on to that of increased effusion, its natural sequence.

Now, if the effusion continue or remain unabsorbed, or is poured out in greater quantities than can be taken up by absorption, it naturally follows that the condition of hydrothorax will be the result; if, on the contrary, the effusion be moderate in quantity, or if absorbed, it as naturally follows that the improvement of the symptoms is but the forerunner of convalescence.

In addition to increasing difficulty of breathing, a difficulty sometimes so great as to call into operation the action of muscles other than those of respiration, extending even to the caudal ones, causing an upward and downward motion of the tail similar to that of a pump handle, the other characteristic signs are dropsical swellings, commencing generally at the sternum, extending along the floor of the abdomen, and finally invading the areolar tissue of the extremities; flapping of the nostrils, protrusion of the head, and increased roundness or bulging of the ribs, and the hair of the mane and tail is easily pulled out. In some instances of hydrothorax the legs remain unnaturally fine, even to the fatal termination.

It may be mentioned that the appetite is capricious, and that the symptoms generally are subject to variations—the diagnostic ones being those detected by percussion and auscultation.

## TREATMENT.

The treatment of pleurisy in its acute stage differs but little from that of pneumonia, unless, indeed, the pleuritic pain be acute, then opium is essentially necessary. In the very earliest stage of the inflammation, when the pain is usually most acute, a dose of tincture of opium, in combination with a pint of linseed oil, has usually a very decided anodyne effect, particularly if its soothing effect be assisted by warm fomentations to the affected side. The opium may be repeated if the pain continues; usually, however, the acute pain subsides in a few hours; and, provided the pulse remain hard, aconite may take the place of the opium. If the pulse be soft and showing little irritation, but little treatment, beyond warm fomentations to the sides, clothing the animal warmly, allowing it to drink freely of cold water, in which an ounce of potash nitrate is daily dissolved, and supplying it with nutritious, easily digestible food, is necessary. The fomentations to the sides are not, however, to be neglected, so long as the breathing remains at all laborious; experience having taught me that they not only soothe the irritation and pain, but that they most materially promote convalescence, and diminish the tendency to hydrothorax and death, but they should not be applied for more than half an hour at a time; their too long-continued application often becomes annoying to the animal and causes restlessness.

Bleeding, although often indicated by the hardness of the pulse, seldom does any good; it certainly has no effect on the duration of the disease, even in cases where its primary effects have seemed to afford relief to the pain and respiratory embarrassment; and in the majority of instances where it has been indiscriminately performed, it has delayed the convalescence, increased the tendency to hydrothorax, and increased the rate of mortality.

*Blisters.*—Of the application of the so-called counter-irritants, I can only repeat what I have taught, that with rare exceptions they are injurious. Veterinarians, however, generally maintain otherwise; and in support of their arguments appeal to the practice of physicians, who, with rare exceptions, until lately practised this method of treatment. Within the last few years opinion has greatly changed; and in the latest book on medicine, namely, “Reynolds,”\*Dr. Austin, article *Pleurisy*, page 942,

vol. iii., says—"The treatment by so-called 'counter-irritation,' as pursued by many physicians, is no less repugnant to me than is that by mercury or bleeding. Let me make two admissions:—In the first place, the mere application of a mild mustard plaster, or, still better, of a hot poultice or epithem, undoubtedly may give some ease, perhaps even arrest incipient inflammation; and the use of 'small flying' blisters, in the limited attacks of pleurisy, which are so common in phthisis, undoubtedly appears to give relief in many cases. But the use of large blisters, especially if kept open, appears to me both useless and often prejudicial." I need not here repeat what must be already self-evident, that blisters, especially those extensive ones ordinarily applied, only increase the pain, the embarrassment of breathing, the general febrile condition, and the tendency to excessive effusion and exudation. There are instances, however, in which the application of a moderate mustard liniment proves beneficial; these are the cases in which the disease makes but little progress towards recovery, in which the symptoms remain without much alteration for better or worse for some days. This condition is characterised by languor, prostration of strength, a pulse standing about eighty, with scanty secretion of high-coloured urine, a continued elevation of the bodily temperature, capriciousness of the appetite, and a respiratory embarrassment.

From the tenth to the fourteenth day after attack, the above symptoms being present, the application of the mustard liniment, and succeeding warm fomentations to the sides, promotes the absorption of exuded materials, stimulates the system generally, and increases the excretion of effete materials from the economy, evidenced by diuresis, improvement of pulse and respiratory movement, and a rapidity of convalescence, which otherwise could not have been established. In thus applying the cutaneous stimulants care must be taken that their effects do not exceed that of stimulation or gentle irritation; if the application be sufficiently strong to induce soreness of the sides, it is calculated to do harm, not only by increasing the pain, but also by exaggerating the adynamic or typhoid condition. In no instance where resolution is progressing satisfactorily should this treatment be applied.

The colchicum, as recommended for pneumonia, may advantageously be administered in combination with iodide of

potassium, or the vegetable or mineral tonics, as the case may be, when absorption and excretion of the inflammatory products are not progressing satisfactorily. When debility and anæmia are associated symptoms, I have found the salts of iron, more especially the tincture of the terchloride, to have a marked effect in promoting the absorption of the inflammatory products, fluid and solid. I have a far higher opinion of the effects of iron salts than of iodine or iodide of potassium. The practitioner is, however, at liberty to combine the iodine with the iron.

*Paracentesis Thoracis.*—In all cases where effusion is excessive, no time should be lost in giving relief to the symptoms of distress by the operation of tapping. I do not mean to advocate early tapping in cases of effusion, being strongly of opinion that absorption of the fluid is calculated to save more lives than its evacuation; but when danger to life is imminent from its excessive quantity, the operation should never be delayed until prostration and debility are associated with the dyspnœa. The most successful method of performing paracentesis thoracis is by means of an instrument, consisting of a trocar and cannula, guarded by a tap, by which a small quantity of the fluid is withdrawn, the exact nature of which can be identified. If fluid be present it can be withdrawn, an elastic pipe being attached to the tap, and the serum allowed to flow into water. Should the fluid be purulent or too thick to flow through the cannula, a suction syringe is to be applied, the force of which is to be sufficiently strong to cause its evacuation.

Dr. Bowditch's rules for performing this operation are as follows:—"Find the inferior limit of the sound lung behind, and tap two inches higher than this on the pleuritic side, at a point in a line let fall perpendicularly from the angle of the scapula. Push in the intercostal space here with the point of the finger, and plunge the trocar quickly in at the depressed part; be sure to puncture rapidly, and to a sufficient depth, as you may be balked by the false membranes occluding the cannula.

"It will sometimes happen that with the greatest care and trouble we are unable to get a flow of fluid at the part where we first puncture; it is then our duty to try elsewhere, for our failing may be owing to unusual thickness of the false membranes in the lowest inch or two of the pleural cavity. We thereupon repeat the puncture a little higher up, and further

towards the axillary line, and here we perhaps find fluid; at any rate, no harm has been done by the two punctures.

"The circumstances under which paracentesis ought to be performed for pleurisy are the following:—

"1st. In all cases of pleurisy, at whatever date, where the fluid is so copious as to fill one pleura, and begins to compress the lung of the other side; for in all such cases there is the possibility of sudden and fatal orthopnoea.

"2d. In all cases of double pleurisy, when the total fluid may be said to occupy a space equal to half the united dimensions of the two pleural cavities.

"3d. In all cases where, the effusion being large, there have been one or more fits of orthopnoea.

"4th. In all cases where the contained fluid can be suspected to be pus, an exploratory puncture must be made; if purulent, the fluid must be let out.

"5th. In all cases where a pleuritic effusion occupying as much as half of one pleural cavity has existed so long as one month, and shows no sign of progressive absorption."

In our patients the puncture can be made in the fifth or sixth intercostal space, and as near to the anterior margin of the rib as possible. It is usual to divide the skin with the lancet, and to stretch it before introducing the trocar, so that, when the fluid is withdrawn, the external wound is not directly opposed to that in the tissues. By this method admission of air into the cavity of the thorax is prevented.

Physicians are agreed that it is not necessary to extract the whole of the fluid, and that the removal of just so much as may be necessary to relieve substantially the mechanical distress will, in most cases, give the necessary spur to the natural process of absorption, by means of which the rest of the fluid will be taken up. Should the cannula become blocked up by fibrinous coagula, and the escape of the fluid thus prevented, a whalebone probe must be introduced.

After the operation the animal's strength must be maintained by good food and stimulating tonics, such as *nux vomica*, ferrous sulphate in combination with hydro-muriatic acid. If the animal does not feed, it should be supplied with milk and eggs instead of its drinking water for a day or so; but, for reasons already given, all such food must be partaken by, not forced on, the animal.

## CHAPTER LXIV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (K.) DISEASES OF THE HEART AND ITS MEMBRANES.

THE weight of the heart of the horse varies according to the animal's weight and size—from 8 to 20 lbs; the average, however, is about 10 lbs. In the ox, sheep, and pig the weight of the heart is to that of the weight of the body as 1 in 220, and in the dog as 1 in 90. Anything much above this average is indicative of hypertrophy.

Heart diseases, although rare in the lower animals compared with their frequency in man, and much more difficult of diagnosis, are neither infrequent nor unimportant.

For the convenience of description they may be arranged under two heads, namely—1st. Diseases of the heart and valves; 2d. Diseases of the cardiac membranes.

1st. Diseases of the heart are divided into functional and organic.

(a.) *Functional derangement of the heart*, characterised by palpitations, irregularity, or intermittence of the pulse, may arise from debility, indigestion, blood poisoning, as in purpura, hæmo-albuminuria, epizootic catarrhal fever, &c.

*Palpitation* of the heart is often due to dyspepsia, disappearing with the indigestion and reappearing with another attack, both in the horse and dog. It also arises from nervousness, and if a nervous animal be approached rapidly and roughly, the beatings of the heart are often distinctly heard; and if the pulse be felt, it may be irregular and intermitting. If one were immediately to form a diagnosis that these results were due to heart disease, a few minutes' further observation would point out the error.

Palpitations and irregularities are symptomatic of other disorders, when they occur occasionally only, and when other signs

of heart disease are absent ; if persistent, they indicate, separately or conjointly, some organic lesion of the heart or its membranes. Repeated examinations of the patients are necessary before forming a conclusion.

(b.) *Organic Diseases*.—Carditis, or inflammation of the substance of the heart, is always circumscribed ; a general or diffused inflammation, which would be immediately fatal in consequence of destruction of function, is fortunately never witnessed. Partial or circumscribed carditis, unless it be due to injuries, and associated with exocardial effusion and exudation, presents no symptoms during life, and is only discoverable *post mortem* by the presence of a small abscess or circumscribed deposits of lymph ; but although the cardiac walls are not subject to inflammation, they undergo various alterations inimical to the well-being of the animal, and may be described as follows.

1. *Hypertrophy*.—This may be of three kinds:—

(1.) *Simple hypertrophy*, where the walls are merely thickened without alteration of the cavity.

(2.) *Eccentric hypertrophy*, where the walls are thickened and the cavities enlarged.

(3.) *Concentric hypertrophy*, where the walls are thickened and the cavities diminished.

These conditions are manifested by a more or less persistent palpitation or increased cardiac force, the cardiac sounds being often heard upon both sides of the chest. They are due to some obstruction to the circulation consequent upon chronic pulmonary disease, constriction of some of the large vessels, and are best combated by a moderate diet, freedom from excitement, and, if depending upon broken wind or any impediment to the pulmonary circulation, by small doses of arsenic.

The most common organic change which occurs in the hearts of horses and dogs is that due to atrophy and fatty degeneration. This change is generally met with in aged subjects, and in those which have been pampered and irregularly exercised. It is also a result of pericarditis, and as a sequel to epizootic diseases in which the pericardium has been involved. It is also induced by blood affections, as purpura, azoturia, and scarlatina.

During life the progress of atrophy and degeneration is manifested by a more or less slowly increasing debility of the circulation, exemplified by diminished cardiac impulse, irregu-

larity of the pulse, and a tendency to cedema of the extremities, with difficulty or inability on the part of the animal to perform ordinary labour.

I have repeatedly watched the progress of this disease, and have found that when the animal is near death there has been capriciousness of the appetite, extreme muscular debility, a peculiar rusty-red appearance of the visible mucous membranes, and a want of correspondence between the pulse and the contraction of the heart. The cardiac sounds have been often loud, amounting to palpitation, but the impulse has been feeble, and the pulse weak and irregular, arising from the fact that the feeble cardiac impulse is not transmitted.

In hypertrophy of the heart, more particularly that of the left ventricle, when the walls are increased in thickness, and the cardiac contractions proportionately strong, the blood is propelled into the arteries with increased force, and the pulse is strong and hard, so long as the circulation is unimpeded by aortic obstruction; but when the heart is atrophied, or its walls attenuated, and its cavities enlarged, the pulse will be of the opposite character—soft, weak, and irregular. If the hypertrophied heart's action be intermitting, the strong pulse will be intermitting also; but in atrophy the pulse will be intermitting and irregular, even providing that the beatings of the heart be regular in their succession.

The *post mortem* examinations reveal the heart apparently enlarged, but the enlargement is due to dilatation of its cavities and attenuation of its walls. The muscular structure of the whole organ presents a pale or fawn-coloured appearance, is soft to the touch; and when examined microscopically, the most noticeable change is the absence of the transverse striæ of the muscular fibres, with here and there true fatty degeneration. In the dog, atrophy of the muscular fibres is generally due to fatty infiltration, which, pressing upon the muscular fibres, cause their removal by absorption. But in the horse the change is more commonly found in the sarcous elements themselves, by which they first lose their truly muscular characteristics and their power of contractility, and finally become converted into a fatty material.

I cannot recommend any treatment calculated to arrest the progress of this change in the horse, but would suggest that the chlorate of potash (which has the power of arresting some



degenerations) might be administered. For the dog, mild purgatives, regular exercise, and light food constitute the best treatment, for, as already pointed out, the atrophy is generally simple, except in old dogs—depending upon the pressure of the fatty infiltration.

#### PERICARDITIS.

Inflammation of the pericardium is often associated with epizootic diseases. In the ox it frequently arises traumatically.

When not arising from injury, its causes are similar to those of pulmonary diseases and rheumatism, with which it is often associated. There are but few instances of epizootic pleuro-pneumonia in the horse but are complicated with inflammation of the pericardium; indeed, during some seasons, epizootic diseases partake of the rheumatic type, and involve not only the pleura, pericardium, and endocardium, but also the fibrous structures of other parts of the body.

*Symptoms.*—In addition to general signs of fever, which is often of a severe type, there is a peculiar irritability and hardness of the pulse, indicative of irritability of the heart; the pulse is a short, angry beat, and the heart's action is often irregular; sometimes bounding and violent, at other times feeble and fluttering. There is coldness of the extremities, and acceleration of the respiratory movements.

Leblanc, who is followed by Percivall, Gamgee, and others, says that a marked contrast between the violent heart-beats and smallness of the pulse are characteristic signs of endocarditis, and that they are very important in distinguishing endocarditis from pericarditis. I cannot confirm this conclusion, having repeatedly witnessed these symptoms in pericarditis unassociated with disease of the endocardium. Generally, however, both membranes are implicated in the inflammation, and it is a most difficult matter, in the lower animals at least, to distinguish between the two affections.

Associated with this inflammation, spasms or cramps of the superficial muscles are often witnessed. Most frequently the cramps are confined to the muscles of the pectoral region and neck; but occasionally those of the posterior extremities are also more or less violently affected. The physical sign of pericarditis is a to-and-fro friction sound, resembling a rasping murmur,

synchronous with the cardiac movements. This sign is subject to variety in tone and degree, depending upon the nature and extent of the exudation or effusion. If much fluid be present, the sound becomes lost, being replaced by a dull, churning noise; as the effusion diminishes by absorption, the to-and-fro friction sound returns, unless, indeed, the pericardium has become generally adherent to the heart's surface. If this sound be associated with a deep-seated blowing or bellows murmur, which sometimes begins to be distinguished when the other ceases, or, in the words of Watson, appears to supervene upon it or to take its place, the combination of sounds indicates an affection of both the external and internal membranes—pericarditis and endocarditis. Dr. Stokes says, "that in respect of morbid anatomy, cases of pericarditis may be arranged into three classes. In the first class are to be placed those in which there is a slight though general effusion of coagulable lymph. In the second, those in which there is superadded the secretion of serum in abundance, causing distension of the pericardial sac. In the third class are to be placed those cases in which signs of muscular excitement, if not of myocarditis, are added to the preceding conditions.

"As the disease advances from the first to the last of these forms, there is progressive increase in the violence of the inflammation, denoted in the second form by the occurrence of excessive serous effusion, and in the last by the altered and impaired condition of the heart itself. Death tends to occur by syncope, induced by paralysis of the left ventricle. The muscular substance of the heart is paralyzed, being of a dirty brown or yellow colour, flabby, and easily torn—a condition which speedily leads to passive dilatation of the heart, general cachexia, and dropsy."

*Post mortem* examinations, as conducted in the dissecting rooms, go to prove that pericarditis, as evidenced by alterations of structure, is not such a common form of disease as endocarditis. In the human being it is stated that about thirty-three per cent. of cases examined *post mortem*, varying from the ages of eighteen to thirty-nine, and about seventy-one per cent. from ages between forty-eight and eighty, show white spots in the heart. These white spots are supposed by some pathologists to be due to previous pericarditis, and by others to attrition from rubbing

of the part against the pericardium applied to the sternum, induced by dilatation of the heart or impeded action of the lungs. These white spots are seldom met with in the pericardium of the horse, whilst they are not uncommon on the endocardial surface.

*Traumatic Pericarditis.*—In ruminants, particularly cattle, foreign bodies often find their way into the pericardium, wounding both it and the heart. Whilst the carditis so induced is circumscribed, and merely surrounds the point of puncture, the pericardial inflammation and exudation involve the whole surfaces of the membrane.

Cattle are exceedingly fond of chewing and swallowing all sorts of substances; for example, nothing seems to give greater pleasure to a cow than to have an old boot or other piece of leather in its mouth, and this it will chew at with evident gratification. An old brush is also a dainty morsel, and I have seen as a consequence of this that the pericardium has been pierced by the brush nail. Many kinds of sharp-pointed materials have been found in the pericardium of cows. I have seen hair-pins, horse-nails, needles, table-knife, iron wire, &c. &c.; and, as a matter of fact, the smarter the maids are in a dairy the greater the danger of this disease amongst the cattle, for smart dairymaids use numerous hair-pins, some of which become lost amongst the food and are swallowed by the cows.

In some instances the foreign body is gradually forced from the chest into the thoracic walls, as proved by the following three cases related to me by Mr. Malcolm Walker, V.S., Alexandria, Dumbartonshire, in a letter dated August 1888:—

“The first was an Ayrshire milch cow, said to be off her feed for a few days. On examining her, I found the pulse weak and irregular, the rumen impacted and flatulent. I ordered a tonic ball to be given three times a day, the ball containing ginger  $\mathfrak{z}\text{i}$ ., carb. ammonia  $\mathfrak{z}\text{ii}$ ., made up with treacle. In two days she seemed better; she had been ruminating occasionally, the pulse still weak, irregular, and an anxious look about her. I saw her again in two days, when she appeared much worse; the nose poked out, small, weak, irregular pulse; laborious breathing, dropsical swelling below the sternum; the sounds of the

heart almost inaudible. I then diagnosed it traumatic disease of the heart, ordered tonics, stimulants, and good nourishment. The swelling on the chest increased to a considerable size. I punctured it in several places, and it gradually diminished; the appetite slowly returned, and the cow seemed to improve every day and get all right again for a few weeks; but she still had the anxious look, and a grunt when she moved about. I was called to see her again in about four weeks after the first attack; the owner said she was very lame in the off fore leg. I had her taken out of the byre, which was dark, and found her lame, caused by a swelling on the off side of the chest, immediately above the sternum and close to the point of the elbow. This swelling being hard, and very painful to the touch, I suspected the presence of some foreign body. I made a free incision into it and introduced my finger, and felt the sharp point of a needle. I enlarged the wound so as to admit my finger and thumb, then I got hold of a very large darning-needle and took it out; it was the largest needle of the sort I ever saw,—it was 5 inches long. I treated the wound with fomentations and carbolic dressings for about two weeks, then I had it thoroughly washed and examined, and found a small dark-coloured cord, which came away with a gentle pull. As long as this cord remained in the wound there was a very offensive smell from it, but very soon after it was removed the wound healed up and the cow did well.

“The second case was in a milch cow, that had been treated four weeks by another practitioner for indigestion. I at once suspected some chest disease, from the grunt she had, and her thin, emaciated appearance. I went to the left side of the chest to auscultate, when my cheek came in contact with a very painful nodule, about the size of an egg. I examined it carefully, and felt something hard in its centre. I cut down on this hard spot, and came on the point of a needle, which I removed with the forceps, it being over the average size of darning-needles too ( $4\frac{1}{2}$  inches). The grunt immediately left her, and the appetite improved every day. I dressed the wound the same as in the first case, and removed the cord in three weeks. The wound healed quickly, and the cow is now as well as ever she was.

“The third case came under my notice on the 1st of this month (August). This cow had been treated by another practitioner for some days (for what I don't know). The owner told me she had been off her feeding and wasting away for some time. I observed a swelling on the left side of the chest, above the sternum, and close to the point of the elbow. This swelling was also very painful to the touch; it was about the size of my two hands. After manipulating it carefully, I could feel the hard nucleus that I felt in the others. I cut down on it, and got hold of a darning-needle of a smaller size, and

half of the eye broken off. I ordered the wound to be treated with fomentations and dressed with carbolic liniment. I have not seen the cow since, but the owner writes me stating that she appears all right, and feeding well. I expect to see her about the end of this week to remove the cord, as in the others.

"I may mention that all these wounds, after the needles were removed up to the time the cord or slough was taken away, had a very offensive smell."

Traumatic pericarditis is generally manifested very insidiously, diagnostic symptoms being often absent for long periods after the foreign body has been swallowed, no symptoms being present, so long as it remains in the digestive cavity, nor during its course to the heart, until it materially interferes with the functions of that organ. I have repeatedly observed instances where cows have presented no other sign of disease than an occasional attack of flatulence or indigestion, and have been sleek and well-doing, until the act of parturition, when they have rapidly succumbed, and the *post mortem* has revealed the pericardium immensely thickened, adherent at its surfaces, and the exudate in a condition of organisation, sometimes of a consistence resembling cartilage; the adhesions between the reticulum, diaphragm, and mediastinum and the walls of the canal being so organised as to leave no doubt of the lengthened period since the injury had been inflicted; the act of parturition, more especially the contractions of the abdominal muscles, having evidently altered the position of the hitherto quiescent foreign body, and thus excited a fresh attack of inflammation. More commonly, however, the symptoms of the lesion have become gradually diagnostic; at first symptomatic of indigestion, with capriciousness of the appetite, flatulence, and eructation of gases, and gradual emaciation. After a while, the pulse becomes exceedingly small; the jugular veins are distended; there is also a well-marked jugular thrill or pulse, extending even as high as the bifurcation of those veins, associated sometimes with palpitation of the heart. To these succeed œdema of the intermaxillary areolar tissue, gradually extending down the neck to the dew-lap; in some instances clonic spasms of the superficial, particularly the cervical muscles. Continental writers state that the diagnostic symptoms of traumatic from simple pericarditis

are a loud gurgling cardiac sound and eructation of gases. The gurgling sound is supposed to arise from the greater consistency of the pericardial effusion, and its admixture with various gases. The eructations are supposed to arise from the communication between the pericardium and stomach, allowing the gas formed in the former to pass into the latter, and thence into the mouth. If these latter symptoms were constant, their importance and value would be beyond doubt. I have, however, seen cases in which neither eructations, gurglings, nor splashing were detectable, and in 1857 I reported two cases in the *Veterinarian*, in which absence of all cardiac sounds was mentioned, this absence of sound being ascribed by me at the time to the thickness and plasticity of the exudate rendering all sounds inaudible.

In some instances there is a painful cough and disturbance of the respiratory movements; in other cases cough is absent, and the respiratory movements unaffected, or even slower than natural. The *post mortem* examination tends to prove that the pericardial exudations are of varying ages, and that the animal has suffered from repeated attacks of pericarditis. This conclusion is arrived at by the fact that two or three, or even four layers of exudation, more or less separable one from the other, are found; the older exudate presenting the appearance of a corneous change, being in consistence similar to cartilage, and not easily cut with a knife; then another layer of exudation of apparently more recent origin; then another layer or two of more recent origin still, all being more or less tinged with the chemically altered metal. In some instances the pericardial surfaces are intimately adherent one to another; in others they are here and there connected loosely by bands of lymph, separated from each other by a turbid fluid, whilst in others the exudate is prevented from becoming adherent by the interposition of fluid. It is, however, more or less vascular, and gradually assumes the appearance of a villous membrane.

Unless the foreign body can be removed as in Mr. Walker's cases, it is obvious that no treatment can be suggested for this form of pericarditis, and it is advisable to make the best of the animal before emaciation renders it worthless. For the prevention of this, attendants on cattle should be enjoined to see that the food be free from any of the above-named foreign bodies. When cattle are attended by females the danger is greater from the pins, &c. worn in the dress becoming mixed with the food.

The treatment of idiopathic pericarditis must be directed to allay pain and undue irritability. For this end aconite is recommended; if pain be great, repeated doses of opium are to be administered; the bowels are to be kept regular by moderate doses of oil, and the absorption of the effusion promoted by diuretics. It was supposed at one time that calomel had the power of causing the removal of the exudate, and it was consequently largely employed; but its administration is now generally condemned, and Sir Thomas Watson confesses, in the following remarkable words, that the hope which he once cherished that the inflammation could be controlled by the constitutional influence of mercury has faded away. He says—"Pericarditis has been known, not seldom, to spring up while the patient was already under mercurial salivation. I am obliged therefore to recant the advice which I was formerly in the habit of giving in respect of mercury as a remedy for acute pericardial inflammation." If debility be present, the weakened heart must be supported and invigorated by moderate doses of stimulants in combination with opium. Bleeding, except to relieve urgent symptoms in the earlier stages of the disease, had better be withheld, as there is a strong tendency to an early diminution of the cardiac energy. Blisters are not called for in the earlier stages of the disease, but their application may, in some rare instances, be necessary to promote the absorption of the effusion. Tonics, more especially the salts of iron, prove useful in promoting the absorption of the effused fluids, and are to be given alternately or in combination with diuretics or the iodide of potassium. In the rheumatic form colchicum is indicated. Digitalis, so highly recommended by some authors, appears to me to act injuriously; it destroys the appetite, is uncertain in its action on the heart, and, if persevered in, its toxic, cumulative effects are apt to cause serious derangement.

To sum up, it may be stated that warm fomentations to the side, warm clothing, bandages to the legs, with careful administration of remedies calculated to relieve such urgent symptoms as may arise during the progress of the disease, and allowing the animal a plentiful but not over-abundant supply of easily digestible nutritious food, are the general principles of safe treatment. If there be danger of death from hydrops pericardii manifested by orthopnoea, obstruction to the venous

circulation, and a serious interference with the heart's action, paracentesis is to be performed; a small trocar being used, which is to be introduced carefully at the side of the sternum, between the cartilages of the fifth and sixth ribs.

#### ENDOCARDITIS.

As a sequel to articular rheumatism, endocarditis is a more frequent form of disease than the last described one. It is an inflammation of the membrane lining the cavities of the heart, and presents symptoms similar to those of pericarditis; the difference being that in its purity the blowing sound—the “bellows murmur” already referred to—takes the place of the to-and-fro friction sound. The marked venous pulse, and the want of correspondence between the pulse and the cardiac impulse, laid down as diagnostic of this disease by Leblanc and Gamgee, are seen in other cardiac affections, and are of no diagnostic value. I am, however, of opinion that the clonic spasms of the superficial muscles, already mentioned, along with hurried breathing and a tendency to syncope, if the head be suddenly elevated, or the animal in any way disturbed, are more marked in this than in any other cardiac affection. It is very true that in cardiac degeneration there is a tendency to syncope from debility of the circulation. This condition, however, differs from acute endocarditis by the absence of febrile disturbance. Endocarditis is much more fatal than any other acute cardiac affection, for the reasons that it is often associated with a mal-condition of the blood; that it leads to valvular alterations, and to a deposition of fibrinous coagula on the valves, which destroy life by interfering with the circulation of the blood, or, carried away by the blood to other parts of the body, by obliterating the capillaries of other organs, leading to softening, abscesses, or sudden death, or undergoing degradation by poisoning the circulatory fluid.

*Morbid Anatomy.*—The first effect of inflammation of endocardium is seen in the form of red spots, streaks, and patches. The redness is always most intense in the neighbourhood of the valves, which in some instances lose their integrity, and become ruptured or detached from their tendinous cords. Supervening upon this, lymph is exuded both into the substance of the



membrane and upon its free surface. That upon the free surface is often washed away by the current, but, generally speaking, it is found between the folds and upon the free surfaces, constituting warty excrescences upon the valves.

In a specimen now before me, obtained from a cow which had three months prior to death suffered from rheumatic arthritis, there are no less than five-and-twenty of these excrescences, more or less organised, attached to the surfaces of the tricuspid valve. They vary in size from a pin head up to a large nut; some of them being in the auricle and some in the ventricle, attached not only to the valves but to the chordæ tendinæ, carnæ columnæ, and musculi pectinati, whilst the spaces between the tendinous cords are filled with coagulated blood. In the left side of the heart there are also traces of deposition within the mitral valve, having the shape of irregular white spots. It will be thus seen that there is not only a deposition upon the free surfaces, but also an exudation in the membrane itself. In some instances the valves become ulcerated, and even the cardiac walls perforated, establishing a communication between the two cavities.

The growths of endocarditis differ from those clots or coagula which form during or after the agony of death, and they must not be confounded with the corpora aurantii, which are generally more or less enlarged in aged subjects. The coagula, which form during or after death, are not adherent to the parietes, are soft and easily removed.

In the treatment of endocarditis particular care must be taken to pursue no treatment calculated to lower the heart's action, as debility of the circulation greatly favours the tendency to coagulation of the blood in the heart, and the consequent formation of these coagula. For the same reason, remedies which have the power of modifying the coagulation of the blood, such as the nitrate of potash, or, when debility is present, the bicarbonate of ammonia, are to be prescribed; and for the reason that inflammations, artificial and natural, increase the fibrinous condition of the blood and its tendency to coagulation, blisters, setons, and all other remedies which constitute the so-called counter-irritant treatment, are to be avoided. The treatment recommended for pericarditis is applicable for endocarditis, with this exception, that those sedatives which diminish the cardiac energy are to be

carefully and cautiously administered. They are certainly useful, more particularly aconite, in relieving disquietude and irritability; but they should be given in small doses only, and for the reason above stated, that all remedies calculated to lower the action of the heart promote the tendency to fibrinous coagulation.

The results of endocarditis are various diseases or alterations in the form and structure of the valves, leading to ulceration, perforation, pulmonary apoplexy, and ultimately to the death of the animal.

#### DISEASES OF THE VALVES.

For various reasons, such as the distance of the heart from the thick and muscular thoracic walls, valvular diseases are exceedingly difficult of diagnosis. They are generally due to a change of structure, caused by endocarditis, mechanical rupture, or morbid growths. The result is imperfect valvular action, indicated by difficulty of breathing when the animal is subjected to exercise, the venous or jugular pulse, the so-called vertigo or megrims, sometimes a tendency to œdema of the limbs, and modifications of the cardiac sounds. These modifications are as follows:—A bellows murmur with the first sound indicates mitral disease or insufficiency; a bellows murmur with the second sound indicates aortic insufficiency. Beyond this my experience does not enable me to describe more definitely any train of symptoms which are of diagnostic value. Indeed, I have repeatedly found the cardiac sounds modified, a distinct venous pulse, and irregularity of the heart's action in various conditions unassociated with valvular diseases. For example, the jugular pulse, which would lead one to conclude that the regurgitations were due to tricuspid incompetency, is generally present in various alterations of the cardiac walls, and in pericarditis, whether it results idiopathically or traumatically. Practically it may be stated that an animal suffering chronically from the above symptoms is only fit for the slowest kind of work.

Ulcerative and warty endocarditis are said by Ziegler to differ only in the degree of their intensity. In the one there is a growth of white fibrous and elastic tissue in the valvular reduplications, which is sometimes absorbed; at other times the valves become thickened, form abnormal adhesions, and

contract the orifices. Fibrinous exudates form on the surfaces of the valves, and these, when carried away, are conveyed to the lungs, if on the right side, or to the brain, kidneys, &c. when on the left side, inducing embolism. Warty excrescences on the valves, observed in cases of swine fever, have been supposed to be the result of or associated with that disease; but according to Mr. Cope (Report of Board of Agriculture, 1894), out of 3065 hearts taken from suspected cases of swine fever, 1039 were found to have that disease, and out of this number 270 showed disease of the valves, but of these only 107 were associated with swine fever. This shows that disease of the valves in the pig is induced by other causes than as well as swine fever.

It appears that the slightest wound on the surface of the valves is followed by a fatal endocarditis, said to be induced by micro-organisms, which are harmless in the normal circulatory apparatus. The *Staphylococcus aureus*, *Streptococcus pyogenes*, and the cocci of septicæmia have induced similar conditions.

#### ANGINA PECTORIS.

The condition termed angina pectoris, or breast-pang, a disease characterised by agonizing pain and distress in the human being, undescribed, so far as I am aware, in the lower animals, is a form of disease which I believe sometimes affects the horse. This disease, in man, is defined as a pain or spasm referable to the lower part of the sternum, extending to the left scapula and root of the neck, and manifesting itself in paroxysms of great severity. I feel confident that in one horse I have seen this condition in a well-defined form. The subject in question was an aged cart-horse brought for my examination in the year 1873. For twelve months previous the horse had done but little work, owing to the fact that when he was excited by work or exercise he manifested the most exquisite agony in the near (left) fore limb, the muscles of which, more particularly the pectorals and those of the neck, became violently convulsed, the limb itself being alternately rigidly fixed by muscular contraction and powerlessly paralyzed, so that the animal was quite unable to use it, and if forced to move he painfully dragged the limb, or sometimes fell to the ground. Whilst at rest the paroxysms seldom occurred,

but I noticed that there was a continual spasmodic twitching of the above-mentioned muscles, as well as a dread on the part of the animal when suddenly approached. On examining the heart and circulation there was a distinct jugular pulse, great irregularity of the heart's action, a loud cooing or blowing sound and strong impulse, indicative of hypertrophy, and such a want of correspondence between the cardiac energy and feeble pulse as to lead me to the conclusion that the horse was suffering from valvular incompetency or impediment, to overcome which, hypertrophy of the cardiac walls had become established.

Angina pectoris is supposed by physicians to be due to dilatation of the cardiac cavities, degeneration of the walls, some condition of the aortic valves which permits the regurgitation of blood into the heart, or some sudden impediment to the coronary circulation. The condition of the horse in question pointed to the conclusion that the cause was due to aortic impediment. Unfortunately I lost sight of the animal, and I am unable to confirm this view by any *post mortem* examination. The latest theories upon the cause of this painful malady are as follows:—

(1.) Neuralgic affection, commencing for the most part in pneumogastric nerve, and spreading in different directions.

(2.) That it is due to such an acceleration of the movements of the blood, by exercise or otherwise, that it arrives at the heart faster than it can be transmitted onwards, and accumulates in its cavities so as painfully to distend them.

(3.) Ossification of the coronary arteries, which, by failing to supply sufficient blood to the cardiac walls, so impairs their strength that the heart is incapable of contracting upon the increased quantity of blood within its cavities, brought about by exertion or excitement.

#### CYANOSIS.

The blue disease described by some veterinary authors, and depending upon non-closure of the foramen ovale, is a condition which is only met with, and that very rarely, in very young animals. It is due to admixture of the venous with the arterial blood in the left cavities of the heart, owing to the foramen of communication existing in foetal life remaining pervious after birth. It is manifested by blueness of the visible mucous

membranes, difficulty in breathing, and coldness of the surface. Animals so affected live but a short time.

#### ECTOPIA CORDIS.

*Misplacement of the Heart.*—The most common form of cardiac misplacement is that in which the heart is situated outside the chest. I have specimens in my possession in which both ventricles and auricles are outside the chest, and in which the heart communicates with the interior of the body through large foramina in the sternum.

## CHAPTER LXV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (L.) AFFECTIONS OF THE DIAPHRAGM.

##### SPASM OF THE DIAPHRAGM,

OFTEN confounded with palpitation of the heart, is generally caused by over-exertion, such as a fast run in the hunting-field; it is also sometimes seen in tetanus. Its most prominent symptom is a convulsive motion or jerking of the whole body, accompanied by a dull, thumping noise, unconnected with the pulsation of the heart, emanating posterior to that organ in the region of the diaphragm. In some instances the impulse of the heart is barely perceptible; the pulse is small and weak, and there is great difficulty in breathing. The spasmodic movements of the diaphragm are not synchronous with the pulse: this, along with the fact that the sound proceeds from parts posterior to the heart, at once points out the difference between this affection and cardiac palpitation.

*Treatment.*—Generally speaking, a good diffusible stimulant, with warm clothing and quietude, is all that is necessary. Should the symptoms continue, opium is to be administered, and if at any time the dyspnoea be great, and the animal in danger of dying from apnoea, it may be necessary to abstract a moderate quantity of blood in order to relieve undue pulmonary congestion.

##### RUPTURE OF THE DIAPHRAGM.

Many cases of this lesion are reported by veterinarians. I am afraid, however, that in most of them the rupture has been *post*

*mortem*, arising from the pressure of the intestines, distended with gases evolved after death. It is stated that the rupture, when occurring during life, is always found in the tendinous portion of the muscle, and that that occurring *post mortem* is in the fleshy part of it. In nearly every subject brought for dissection, if kept unopened until the abdomen has become much distended, the diaphragm is found ruptured. I am not acquainted with any symptoms diagnostic of this lesion, except those of abdominal pain, distressed breathing, and general disturbance. Mr. Robinson of Greenock tells me, however, of a case in which for two days prior to death there was general uneasiness, pawing with the fore feet, and, what might be looked upon as diagnostic, a dark patch of perspiration was seen to be constantly present on the skin opposite the diaphragm on one side. On making a *post mortem* a knuckle of intestine was found in the cavity of the thorax, forced through a round aperture in the diaphragm, opposite the patch of perspiration. Beyond this, I can gather no reliable information from the writings of veterinary authors which would justify me in laying down any rules for its recognition. If I were to do so, what I might state would be calculated more to mislead than to enlighten.

Should the lesion be found *post mortem* in the tendinous portion, or if at any time the gap be stained with extravasated blood, the probabilities are that it occurred during life.

## CHAPTER LXVI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (M.) DISEASES OF THE DIGESTIVE ORGANS.

WITH very rare exceptions, diseases of the digestive apparatus are results of errors in feeding. Some of them have been already discussed under the head of Dietetic Diseases, and need not again be referred to. I may here, however, briefly observe that horses are best kept in health and working condition when fed upon an admixture of food requiring thorough mastication, and that horned cattle are best kept in health when, in addition to the more nutritious aliments, they are freely supplied with food requiring remastication, such as hay, grass, or straw.

Observations on the diseases of digestive organs point to the conclusion that in the horse the intestines are more liable to suffer from disease than the stomach; whilst in the ox and sheep the reverse is the case; and in the dog, consequent upon its power of vomition, the stomach is more rarely disordered than one would be led to expect from the nature of its food, &c.

In the horse the stomach is a simple organ, small in comparison to the size of the animal, and in contrast with the volume of the intestines. It is but slightly called into action during the digestive process, and provided the food be properly masticated and incorporated with the salivary secretions, it is arrested for a short time only in the stomach, but is passed onward into the intestinal canal, where the process of digestion is completed. On this account the intestines are more liable to disease. It is also a remarkable fact that easily digested food, if given over-abundantly, is apt to derange the small intestines, whereas food containing much woody fibre, such as over-ripe hay, more particularly rye-grass, coarse straw, &c., accumulates in the large intestines, and there causes derangement, inflammation, and even paralysis of the intestinal muscular tissue. It is also a fact worthy of notice, that if food be given artificially prepared by boiling or steaming, it is retained in the stomach itself, and if given over-



abundantly, causes distension, inflammation, paralysis, and even rupture. This is accounted for by the circumstance that food thus imperfectly prepared for digestion is retained or imprisoned by the action of the pyloric structures, and thus distends the stomach by its bulk, or by gases evolved by the process of fermentation, which is apt to ensue. By bearing these facts in remembrance, the practitioner will to some extent be able to arrive at a correct idea of the seat of gastric or intestinal disease.

The food of the horse contains an abundant quantity of starchy materials, and the process by which these are rendered soluble commences in the mouth, not only by their admixture with the salivary secretions, but by a chemical change, through which the non-soluble starch is converted into dextrine and grape sugar, and made fit for the action of the intestinal, biliary, and gastric secretions, and for absorption by the vessels of the gastric and intestinal walls. For the purpose of performing this process the horse is provided with twenty-four mill-stones, in the form of molar teeth, which have the power of crushing and triturating the hardest food, and of an extensive system of salivary organs, which secrete—most actively during the process of mastication—a fluid which most effectively blends with, and chemically changes, the food thus triturated. On this account we find that, when horses are sufficiently but not over-abundantly fed with dry food of a proper quality, the stomach rarely suffers from disease. An error in the diet, however, or a sudden change from one kind of food to another, not only deranges the stomach, but the intestinal canal as well.

In the ox and sheep, the large and complicated stomach not only digests, but also prepares the food for digestion. For example, ruminating animals eat and swallow the coarsest food very rapidly, and they are provided with a large receptacle for its retention, in which it undergoes maceration and reduction to smaller particles by a slow churning movement in the rumen and reticulum, which facilitates its trituration during the process of rumination and after-solution by the digestive fluids.

Without entering further into a physiological consideration of the process of digestion, it will be seen that the stomach of the ox much more actively participates in the process of digestion than that of the horse, and that it is thus rendered more liable to disorder in its first and second, as well as its third and fourth compartments, than the simple, single stomach of the horse.

## CHAPTER LXVII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (N.) DISEASES OF THE STOMACH.

##### INDIGESTION WITHOUT ENGORGEMENT.

FROM various causes, such as improper food, the process of dentition, diseases of the teeth causing imperfect mastication, ravenous feeding, the presence of other diseases, debility of the stomach itself resulting from some constitutional predisposition, or from food given at uncertain and rare intervals, a condition of indigestion is induced in the horse. In young animals the same is induced by draughts of cold milk; removal from the dam at too early an age, or, what is commonly the case in some districts, compelling the dam to work shortly after the birth of the offspring, and allowing it to suckle at rare intervals and when the dam is heated.

*Symptoms.*—In the horse the symptoms are, loss of appetite, or depravity and capriciousness of it, manifested by the animal eating at irregular intervals, or having a desire to eat filth—bulimia—with sourness of the mouth, and usually increased thirst; the animal soon becomes hide-bound, has a dry, scurfy skin, there is irregularity of the bowels, and frequent escape of flatus by the anus. If caused by imperfectly masticated food, such as whole oats or coarse hay, these may be found in the fæces. In addition to the above diagnostic symptoms, there may be a dry cough, or irregularity of the pulse, which may be slower or faster than natural; colicky pains may also be present in some cases, occurring more particularly in an hour or two after the animal has partaken of food; whilst in others, fits of giddiness—megrims—and even paralysis occur, the latter condition being not seldom seen in cattle, and very often in dogs.

In the young the above symptoms are more commonly associated with diarrhoea than in the older animal, in which constipation is generally present. The fæces often resemble the colour of the food: for example, if the horse be fed on

dark-coloured hay or clover, the fæces will be dark-coloured also; if, on the contrary, it be fed on oats, the fæces will be light in colour; and in the young animal, when fed on milk, it will often resemble it both in colour and consistence, mixed, however, with large masses of curdled milk, and often very foetid. I have often noticed that when indigestion is induced by clover, the urine is very dark in colour, and deposits a thick, almost brick-coloured sediment. This condition of urine, however, need not cause any apprehension, as it is often seen in the clover-fed animal without any disease being present. The urinary deposit mostly consists of carbonate of lime, the tinge being due to the colouring matter of the food. Indigestion, however, is a fertile source of deposits in the urine, which result from imperfect nutrition of the tissues, or a chemical change in the constituents of the blood-plasma, due to the products being imperfectly prepared, or containing some material unfit for healthy nutrition, as already referred to in the former parts of this work.

*Treatment.*—Carefully inquire into the cause, and remove it. If due to the process of dentition, the presence of unshed crowns of the temporary teeth irritating and wounding the mouth, or to any irregularity of the dental apparatus, these must be attended to according to the directions laid down under their several heads. In all instances where such causes are not in operation, even when the cause cannot be traced to the food, it will be necessary to make some alteration in the diet, and to examine the various alimentary matters in order to detect the offending one if possible.

If diarrhœa be not excessive, and the animal thereby much debilitated, it will be advisable to give a mild aperient or a moderate cathartic. To the young animal a dose of castor or linseed oil; to the older, a moderate dose of aloes, combined with a vegetable bitter, ginger, or gentian. After the laxative has operated, I have found that the bicarbonate of soda, with gentian, or, where the stomach seems much debilitated, nuxvomica, to have a most beneficial effect, not only improving the appetite, and removing the acidity—pyrosis or “heartburn”—which is usually present—manifested by a tendency to lick the walls or other cold or alkaline material—but strengthening and improving the gastric apparatus.

It may be here stated that the mineral tonics, particularly the salts of iron, are inadmissible, and generally do harm in the earlier stages of gastric debility, or indigestion, often destroying what little appetite remains, and becoming combined with sulphuretted hydrogen in the intestines, tinging the fæces a black colour—a certain sign in all instances that the iron is not digested or absorbed into the circulation, where alone it can be beneficial, and that it is doing harm. It is very true that even in health large doses of iron salts tinge the fæces, but moderate doses have not this effect to any great extent. In some cases the alkaline-bitter treatment fails in having the desired effect. When this occurs the mineral acids, particularly the nitro-muriatic, may prove beneficial. In foals and calves pepsine can be administered, as in all probability the indigestion is due to imperfect secretion of the gastric glands; even in the older animal this is often presumably the case, and more especially when the disorder occurs without apparent cause, the same remedy will prove beneficial. It is almost needless to observe that the dieting of the animal is to be carefully conducted, and that pure air, moderate exercise, and good grooming are essentials to good digestion. Occurring in the winter, if the animal be thickly clothed with hair, clipping will act almost magically, restoring the digestion and appetite, which may have been long impaired, notwithstanding remedies, in the course of a few hours. In the cow, chronic indigestion, as exemplified by recurring tympanites and other symptoms similar to those observed in the horse, and when not occurring from recognisable external causes or other diseases, is often due to the presence of some foreign body in the rumen or reticulum, removeable in some instances by the operation of rumenotomy, hereafter to be described. In all chronic indigestions of the cow this operation is recommendable. I have repeatedly performed it successfully, even where no foreign body has been found, the mere removal of the long-retained food having been sufficient to restore the organ to its healthy condition. In young calves, indigestion, associated with convulsive fits, is sometimes due to hair-balls; these after a time become gradually disintegrated by the movements of the stomach, and the symptoms may slowly disappear. In rare instances hair-balls are found in fully grown cattle, and, as in calves, result from the animals licking each other. In calves

the symptoms of urgency are often relieved by stimulants, such as the carbonate of ammonia or turpentine; should the indigestion, however, remain for a considerable period, recourse must be had to the operation already referred to. It may be here mentioned that common salt given in the food promotes digestion in all animals. In the dog indigestion is manifested by frequent retchings or vomitings and fœtor of the breath, and is best treated by a brisk purgative, antacids, and a restricted diet.

**INDIGESTION WITH ENGORGEMENT—IMPACTION OF THE STOMACH—PLENALVIA—GASTRIC TYMPANITES—HOVEN.**

Distension of the stomach may arise from repletion with solid food, or from the evolution of gases arising from solids or liquids contained within it undergoing the process of fermentation, or disengaged from the gastric walls when the stomach is empty, as occurring in conditions of great prostration.

*The causes in the Horse.*—Impaction of the stomach results from the ingestion of food too abundant in quantity, or greedily swallowed and imperfectly masticated. In those parts of the country where the cooking of food for horses is a common custom, it is found that deaths from diseases and lesions of the digestive apparatus are very common. From the reasons already hinted at, namely, that it is necessary for the food to undergo not only the process of trituration by the teeth, but that it requires to be chemically altered by combination with the saliva, it will be understood that food prepared in any other way, as cooking by boiling and steaming, is unfitted to be acted upon by the stomach, and is consequently retained within it—the animal meanwhile continuing to eat—until its walls become distended, paralyzed, or even ruptured.

Some kinds of food, nutritious in themselves, and theoretically calculated to be proper for the horse, are found practically to be highly dangerous. Wheat, for example, which is highly nutritious, containing, according to Sir H. Davy, 955 parts of nutritious matter in 1000, is found to be improper, deranging the stomach, causing purgation, laminitis, and death. Barley again has a similar effect, and for these reasons is found to be an improper article of diet in Britain. Barley is, however, used with great success in India and the East generally. The

consequences of its feeding are not noticeably bad, and where fed naturally, cases of colic are no more frequent than at home. It is used either crushed or parched, the former giving the best results. In fact, the Arabs train their horses for racing on it. It must not be given in such large quantities as oats, and animals should be fed oftener. This latter fact is necessary with all foods. The horse's stomach is arranged to take small quantities at a time, and therefore feeding should be frequent—at least four times, but preferably five times, a day.

When, however, from various circumstances, such as damaged crops, it becomes compulsory to cook the food, it is necessary that it be given with the greatest caution, in small quantities, and at intervals. Bran again, so useful an article when combined with other foods, or as an occasional mash, if given in large quantities, is retained undigested, and induces a condition of repletion which often proves fatal. Some kinds of hay, musty or otherwise damaged, or too ripe previous to being cut, barley and foreign straw, are also common causes of impaction; whilst green foods, particularly when animals are first put upon them, or if given too abundantly, not only induce engorgement, but also undergo fermentation in the stomach, and thus induce tympanitis.

In horned cattle and sheep the same conditions are produced, most commonly by damp grasses, turnip-tops, maltcums, clovers, &c.; indeed it may be stated that various foods, if given over-abundantly, will cause tympanites, but none so speedily as green clover.

*In horned cattle*, tympanites—hoven or blown—arising from retention of food in the rumen, frequently accompanies other diseases. It is wonderful how great a quantity of food is found in the rumen of an animal which has died from a disease which has existed for several days or even weeks. In many instances, where an animal suffering from pleuro-pneumonia has not partaken of any solid food for a period extending perhaps over a fortnight, it has been found that the rumen contains several bucketfuls of alimentary matters. The rumination having been suspended, there has been no true digestion, and the food partaken of prior to the occurrence of the illness has lain, as it were, in a mass in the inert and paralyzed rumen, having a tendency to undergo fermentation, and thus induce tympanites.

An additional cause of retention in the rumen is found in disease of the salivary glands. Fluorens asserted that from the period of feeding to that of rumination there is a constant abundant secretion of saliva, which is constantly swallowed; if this be stopped, the contents of the rumen become hard and unfit for regurgitation. In this way rumination is suspended, and tympanites induced. These observations have been confirmed by Colin, who also found that if the parotid ducts were opened, and the secretion thus prevented from flowing into the mouth, rumination became suspended.

The act of vomition, rarely performed by the horse, occurring only as a symptom of a grave lesion or disease, might be easily performed by ruminants; in fact the regurgitation of the food during the act of rumination indicates this facility, but vomition itself seldom occurs.

It is not my purpose to enter into a physiological discussion upon this matter, but merely to state that I am of opinion that vomition is rendered difficult in the horse by folds of mucous membrane at the cardiac orifice, and that, if from any cause, such as inordinate distension or rupture of the stomach itself, flaccidity, dilatation, or paralysis of the lower end of the oesophagus—the rugæ become unfolded, and the cardiac orifice opened—that vomition can and does occur. The valve of Gurlt, which is described by some authors as a spiral valve at the cardiac opening of the stomach, has no existence.

The true source of the rarity of the act in ruminants is, I think, satisfactorily pointed out by Mr. J. S. Gamgee, and is due to the fact that these animals are nauseated with great difficulty. I have, however, witnessed very forcible vomition in cattle suffering from indigestion, as well as the passive return of food into the mouth, which occurs during the profound coma of parturient apoplexy. Indeed, upon more than one occasion such ingesta have found their way into the trachea and bronchial tubes, and have caused a fatal pneumonia in two or three days after recovery from the parturient disease.

The dog, pig, and cat are easily nauseated by various remedies, and vomit with great facility.

*Symptoms in the Ox and Sheep.*—Tympanites is diagnosed by a swelling on the left side, which may appear during the time the animal is feeding, or shortly after; the breathing is difficult and

laborious, becoming more so as the gas is generated and as the swelling increases. The oppression of the breathing is manifested by the general appearance of the animal; there is expansion of the nostrils, moaning during the expiratory movement, eructations, dribbling of saliva from the mouth, and some degree of uneasiness; rumination is suspended; the bowels soon become constipated, and if the tympanites is extreme, there will be a prominence and wildness of the eye which is characteristic of obstruction to the entrance of air into the lungs.

The moan or grunt, which is heard not only in tympanites, but in various forms of indigestion, even when unaccompanied by distension, is similar to that of pleuro-pneumonia; and on this account indigestion has been mistaken for pleuro-pneumonia, and credit claimed for curing the latter disease when it had no existence. The moan of indigestion is rather more prolonged, and resembles a groan more than the grunt of pleuro-pneumonia. The gases evolved have been found to be composed of carburetted hydrogen, sulphuretted hydrogen, carbonic acid, and in some cases carbonic oxide. Unless relieved, the animal will die, either from pressure of the distended stomach upon the diaphragm, causing suffocation, or from the absorption of noxious gases into the circulation. In some instances, tympanites is chronic, and, as already stated, may depend upon the presence of foreign bodies in the rumen.

*Treatment.*—In very urgent cases, the most effectual treatment is that of puncturing the rumen with a trocar, and allowing the gases to escape through the cannula. The operation is to be performed on the most prominent part of the swelling, and at equal distances from the spine of the ilium, last rib, and transverse processes of the lumbar vertebrae. When the symptoms are not very urgent, tympanites can be relieved by stimulants and stomachics, such as carbonate of ammonia, turpentine, alcoholic preparations, or the vegetable spices, particularly if given in warm ale. After the symptoms of urgency have been removed, cathartics are to be prescribed, such as salts, with croton and aromatics, care being taken that the animal be kept upon a restricted diet for some days after recovery. When the tympanites becomes chronic, stomachic stimulants, particularly nuxvomica, are to be prescribed; but if, along with the attention to dieting, they fail to give relief, it may be necessary to open the rumen, when the cause may be detected and removed.



## IMPACTION OF THE RUMEN WITH SOLID MATTERS.

The symptoms are similar to the above, with the exception that the swelling, resonant when tapped with the fingers in tympanites, is dough-like and pits on pressure. In some instances I have noticed that the pitting remains for a considerable period after the pressure is removed, indicating that the coats of the rumen have lost their muscular tonicity, and that its movement is in abeyance, or entirely lost. I have also noticed that in such cases medicinal remedies do harm until the viscus has been to some extent emptied, and that the best method of doing this is to perform rumenotomy before the powers of life become exhausted.

The best method of performing this operation is as follows:—After securing the animal by the nose, with its right side to the wall, plunge a sharp bistoury into the rumen, commencing the puncture midway between the last rib and the spine of the ilium, and from four to five inches from the points of the transverse processes of the lumbar vertebræ, cut downwards until the wound is large enough to admit the hand.

Some practitioners recommend that a towel or handkerchief be introduced into the wound, in order to prevent the food falling into the peritoneal cavity. I find it much better to place a suture at the lower part of the incision through the lips of the double wound. When this is done, the contents are to be removed by the hand; the parts are then to be thoroughly cleaned; the incision in the stomach to be first stitched up, its edges being turned inwards, so as to get the peritoneal coat into apposition. The best material for the sutures is small catgut or fiddle-string. The external wound may then be closed with a stronger suture of strong waxed twine, over which a stiff pitch plaster it to be applied. Cathartics are to be administered, succeeded by vegetable bitters, more especially *nux vomica*, and the animal is to be carefully fed. In less urgent cases, where the rumen still retains some tone and power of movement, removal of the impaction is to be effected by cathartics and stimulants, the best cathartic for this purpose being an admixture of croton, aloes, and sulphate of magnesia, succeeded by a plentiful supply of fluids, treacle, and an occasional dose of ginger or ammonia. To restore tone to the rumen when debilitated, common salt dissolved in cold water has been recommended.

This can do no harm, and failing other remedies, may be given in tympanites and impaction.

#### IMPACTION OF THE THIRD STOMACH.

Variouly termed fardel-bound, vertigo, maw-bound. This is a very popular disease, some writers ascribing every case of constipation to impaction of the omasum, basing their conclusions upon the fact that the contents of this viscus are in a dry and hard condition when examined after death. But seeing that this is its natural state, and that, when animals have died from what appeared to be obstinate constipation, its contents have been found moister than natural, I have arrived at the conclusion that what is supposed to be impaction of the third stomach is in reality an inflammation of the mucous membrane of the true stomach—abomasitis—or true gastric inflammation. I do not mean, however, to state that the third compartment does not participate in the disorders of the others; but, on the contrary, that disease commencing in the rumen, reticulum, or abomasum, soon involves the third compartment. I shall not, therefore, separate the description of this disease from abomasitis.

In the gastritis of ruminants a highly disturbed condition of the nervous system is a distinguishing symptom, evidenced either by a high state of delirium, coma, or convulsive fits, indicative of disturbance of the brain proper, or by paralysis of the posterior extremities, when the area of the disturbance is limited to the posterior parts of the spinal cord. Paralysis, convulsions, and coma are also frequently seen in dogs when suffering from gastric affections, and the same may be said, but in a less degree, of the horse.

In addition to the above symptoms, the gastritis of ruminants is characterised by more or less diarrhoea, soon succeeded by an apparent obstinate constipation, which, however, is not due to an obstruction by impacted food, but to cessation of the peristaltic action of the intestines, the contents of the stomachs being found generally more or less fluid after death. In many instances the animal strains violently, and passes both blood and mucus, showing that the inflammation has extended into the intestinal canal, and it is said that a hard swelling may be detected on the right side, arising from distension of the third

stomach. In many cases, however, general swelling of the abdomen, tympanites, supervenes early in the disease, and greatly adds to the animal's suffering.

Outbreaks of gastritis prevail in some parts of Scotland during the spring and early summer, and is known by the term "grass staggers;" and Mr. Clark of Coupar-Angus, in a paper read before the Scottish Metropolitan Veterinary Association, July 4th, 1894, has given an able description of this disease after an experience of twenty years in a district where the disease prevails to a remarkable extent. He states that animals of all ages and breeds become attacked when placed in fields favourable to the development of the disease, such fields being generally poor land or good land poorly treated, and which have been sown with too large a portion of rye-grass and too small a quantity of permanent grasses. When the quantity of rye-grass has been diminished, and that of the permanent ones increased, the disease has disappeared from land known to be favourable to its development.

The disease appears in about a month after the cattle have been turned out, and to disappear upon the advent of the white clover, which occurs from the 10th to 15th of June in Mr. Clark's district.

The disease is dangerous on first year's grass, less so on the second, and rarely, if ever, on a third year's crop; but it has been known not to attack on the first, but during the second year's crop.

Artificial feeding seems to diminish and modify attacks, but prevention can only be effectual by the diminution of the quantity of rye-grass sown, increase of the natural ones, and better treatment of the land.

The premonitory symptoms are obscure: the animal loiters about, feeding occasionally, and when lying down there is a flapping or restless movement of the ears and intermittent tremors at the elbow and flank, and any excitement, even at this stage, such as driving from the field, is apt to induce loss of vision. The next symptom is purging, the fæces being black and watery, with entire loss of appetite, failure of milk, grinding of the teeth, accelerated pulse, cold extremities, blindness, but the sense of hearing seems to be very acute. There is no elevation of temperature.

In three days the crisis is reached, when the animal may become intensely excited, bellows fiercely, presses the head against the walls, and has violent tremors; and, if unfastened, scrambles up against the walls, and gives every evidence that it is suffering pain of the most formidable character.

The *post mortem* reveals acute gastro-enteritis, involving the abomasum and small intestines, and in the rapidly fatal cases the contents of the omasum are in a soft and normal condition,—thus doing away with the idea that the disease is due to impaction of the maniplies (omasum); but if the disease has been more prolonged before death or slaughter, it will then be found that the food is hardened and the walls of the viscus covered with small red or congested spots, “which,” Mr. Clark states, and I agree with him, “has given rise to the erroneous opinion that the animal has suffered from obstinate constipation. Such, I feel certain, is not the case, the hardened or dry condition being the result and not the cause of the disease.” Mr. Clark thinks that the disease is induced by an irritant, being also more or less narcotic in character, causing malassimilation of food, and as a result non-nutrition of blood, accompanied by inflammation of the true stomach and intestines, this being the only constant morbid condition found on *post mortem* inspection.

The treatment recommended as the most successful is linseed oil as a purgative, Epsom salts being highly objectionable, and calculated to reduce the chances of recovery. A large percentage of cases are fatal, and bleeding has proved injurious. When brain symptoms set in, benefit can be obtained by the application of cold water to the head.

When the animal is in good condition Mr. Clark advises slaughter, and mentions that some farmers are under the necessity of renting grass parks—permanent pasture—to tide over the dangerous period of May and June.

Professor Dick said he was successful in treating this disease by repeated bleeding, even to faintness, large doses of purgative medicines given in large quantities of gruel, cold water to the head, &c. He, however, considered the disease to be mere impaction. At the present time no one would think of pursuing such an irrational course in cases of acute gastritis; and having the knowledge that the constipation results from loss of function rather than impaction, we will do well to recommend a

course of treatment calculated more to modify that inflammation than to overcome the seeming constipation. For this purpose sedatives, such as aconite or belladonna, with antacids—bicarbonate soda or potash—and one or at most two moderate doses of an oleaginous aperient, with an abundant supply of fluids for the animal to drink, fomentations to the abdomen, and enemas, are to be prescribed. This treatment is much more calculated to save life than the indiscriminate use of powerful cathartics and stimulants. I speak advisedly, having witnessed many animals destroyed by the administration of repeated doses of cathartics in order to overcome constipation in this affection, the parties prescribing not being perhaps aware that an inflamed part loses its function, and that before the function and the peristaltic movement of the bowels can be restored the subsidence of the inflammation is essential, and that such inflammation is much more likely to be increased than diminished when the inflamed tissue is irritated by drastic cathartics or other remedies, which are supposed to rouse up the action of the bowels. If passage of the fæces is not restored in the course of twenty-four hours after the administration of the aperient, it does not follow that it is necessary to repeat it; time must always be allowed in all inflammatory diseases for the inflammation to subside, and for the weakened or debilitated parts gradually to resume their normal functions. The late Professor Strangeways was very successful in the treatment of this affection. At the commencement he gave a dose of oil, with sedatives, and after the febrile symptoms had to some degree subsided, from eight to twelve ounces of sulphate of magnesia, fifteen grains of quinine, and a few drops of sulphuric acid, ample time being allowed before any additional cathartic was given. It was very seldom necessary to repeat the medicine, as the bowels became gradually restored to their natural healthy condition.

*Impaction of the stomach in the dog and cat* are naturally overcome by vomiting, which also occurs sometimes in the pig, but should this not occur, an emetic is to be administered.

The dog suffers from catarrhal inflammation of the stomach—*gastrorrhœa*—induced by improper food, or occasionally by enzootic influences, in which there is a high degree of fever, hot nose, blood-shot eyes, and quick pulse, abdominal pain, constipation, and frequent or almost constant attempts to vomit; a dense

mucus tinged with bile being sometimes thrown up. The most successful treatment consists in allaying the gastric irritation by small doses of hydrocyanic acid and antacids, and when this is effected, gently moving the bowels by a small dose of castor oil. Enemas may, however, be beneficially employed in the earlier stages, and if the abdomen be tender or swollen, fomentations or a warm bath. If the strength seem to fail, the pulse small, &c., stimulants are to be cautiously given. As a rule, however, they do more harm than good, and if the first dose is not succeeded by visible improvement they are to be discontinued.

#### SYMPTOMS OF GASTRIC IMPACTION IN THE HORSE.

Pawing with the fore feet, especially the near one, eructations of gas, sometimes attempts at vomiting, with occasional discharge of saliva from the mouth, some degree of fulness of the abdomen, colicky pains, tremors of the superficial muscles, particularly those in the region of the left shoulder, with partial sweats upon the body, are more particularly the symptoms of this disease.

True gastritis, except from the action of direct irritants, such as poisons, is but seldom seen in the horse; its stomach may be distended even to rupture, without any marks of inflammation being discoverable after death.

When inflammation of the stomach is induced by arsenious acid, the symptoms are great pain with uneasiness, the animal alternately getting up and lying down, tympanitic abdomen, faeces mixed with mucus, the saliva foetid, and its secretion increased, mouth hot, extremities cold, nausea, purging, and great prostration of strength with delirium. When the irritation is caused by the bichloride of mercury, there is, in addition to the above symptoms, a profuse discharge of saliva from the mouth. The antidotes for arsenical poisons are the hydrated sesquioxide of iron, chalk, albumen, or magnesia, and for the mercury salt, white of eggs, the symptoms of irritation being combated by opium, oleaginous purgatives, and demulcents.

#### TREATMENT OF IMPACTION OF THE STOMACH IN THE HORSE.

Aloetic purgatives; if combined with tympanitis, oil, turpentine, or ammonia; enemas, fomentations to the abdomen; care at all times being taken that the animal be prevented from

throwing itself suddenly down and causing a fatal lesion, namely—

#### RUPTURE OF THE STOMACH.

The symptoms are, sudden tremors, particularly of the fore extremities; in many cases extension of the near fore limb, profuse perspiration, great prostration of strength; the animal breathes heavily, staggers in its walk, looks round to the flanks, and is generally seized with symptoms of vomiting, during which the head is suddenly depressed, the nose brought down to the sternum by spasmodic contractions of the inferior cervical muscles, and in many cases there occurs an actual expulsion of food from both the mouth and nostrils.

The value of vomition as a diagnostic symptom of rupture of the stomach is certainly very great. Occurring in rupture of the stomach, it is said to be due to the muscular coat having first given way, thus allowing the mucous membrane to protrude, and the rugæ of the cardiac orifice to become unfolded. This conclusion is true in part only, for vomition will occur in rupture of the colon or other intestine, and in dilatation of the œsophagus. It must, however, be admitted that the act of vomition is much more complete in rupture of the stomach and dilatation of the cardiac orifice than it is in any intestinal lesion. The differential symptoms of rupture of the stomach, which occurs in the great curvature towards its pyloric portion, and a paralyzed condition of the cardiac orifice, are chiefly those manifested by the general condition of the animal. In rupture, prostration and rapid sinking of the animal powers are very extreme, the pulse feeble and fluttering, and death soon closes the scene; whilst in the other condition, though sweats bedew the body, and the animal exhibits extreme agony, the vital powers still remain tolerably strong; the pulse, though frequent, still retains some fulness, and the surface of the body and extremities are never deathly cold as in rupture.

Abdominal pain, as ordinarily manifested by rolling, striking violently at the belly, &c., is not a constant symptom of rupture. In some cases the animal will stand immoveable, breathing heavily, being seemingly afraid to perform any movement which may increase its anguish; in other instances, the ordinary symptoms of abdominal pain, combined even with delirium, are present.

I have observed that all medicinal remedies increase the severity of the symptoms. This is undoubtedly due to their escaping into the peritoneal cavity. Mr. Percivall asks the questions—Can vomiting take place after rupture? Will the real stomach retain any power of ejection? Would ejection of the contents upwards be produced by the abdominal muscles and diaphragm without the aid of the stomach? And replies, “I should very much doubt it. I should rather feel inclined to the opinion that the act of vomiting should be taken as a proof of the entireness of the stomach. At all events,” he says, “we may have rupture happen without vomiting; and, consequently, we must cease to regard that symptom as pathognomonic, though we may justly consider it, in company with others, as one throwing much light upon the nature of the case. Our guides, in the absence of any one infallible pathognomonic sign, must be—the history of the case, the subject of it, the circumstances attending it, the inflated or enlarged condition of the abdomen, the symptoms of colic or gripes ceasing and becoming succeeded by cold sweats and tremors, the pulse, from being quick and small and thready, growing still more frequent, and at length running down and becoming altogether imperceptible; the countenance denoting gloom and despondency of the heaviest character, with or without vomiting.”

This graphic description contains some errors. For example, the enlargement of the abdomen is not generally associated with rupture; on the contrary, the belly in some instances is smaller than natural, the abdominal muscles being rigid and tense from tonic spasm, and the condition of rupture is undoubtedly associated with vomition, for it not unfrequently happens that the animal dies immediately after that act.

In addition to impaction from over-feeding, the stomachs of old horses particularly become ruptured from degeneration of their walls. When this occurs the gastric walls are exceedingly thin, and atrophied for some distance around the breach; and if examined microscopically the tissues are found to have lost their histological character, being reduced to a granular *débris* or an oily material.

There is no treatment for this lesion; and if the veterinary surgeon is satisfied that it has occurred, he will do well to order the animal to be put out of its misery.



## CHAPTER LXVIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS.

##### CONSTIPATION.

THE bowels of some horses are naturally torpid. Constipation, however, may be looked upon more as a symptom than as a disease in itself. So long as the animal remains in health there is no necessity to employ active remedies for the removal of constipation, and all that is necessary is to give an occasional bran or linseed mash. Should the condition be caused by the nature of the food, such food must be changed, and one of a more laxative description substituted.

In many diseases constipation results from debility of the bowels, and is to be overcome by remedies, the action of which on the healthy body may be considered astringent in virtue of their tonic effects, such as the salts of iron, cinchona bark, gentian, nux vomica, and other tonics and bitters.

Constipation may also arise from paralysis of some portion of the intestines, and if large and repeated doses of cathartics be administered, a fatal termination may be looked for, either from their toxic effects upon the system generally, or their direct irritation on the intestinal canal. It is, therefore, advisable to act cautiously in all cases of constipation, to allow plenty of time for the paralyzed bowel to regain its tone, to rouse it by stimulants and nervine tonics, and to administer enemas, which may contain turpentine. One symptom of paralysis of the bowels is diagnostic, namely, the absence of *intestinal murmurs*. Another may be mentioned of not infrequent occurrence,

especially if the paralysis be in the large intestines, namely, a dilated, dry, and non-contractile condition of the rectum, which feels, when the hand is introduced, as a large cavity with passive walls.

### COLIC.

Colic is of two kinds, namely, 1st. Spasmodic; and 2d. Flatulent.

*Spasmodic Colic.*—A spasmodic contraction of the muscular coats of the intestines, which may run on to inflammation, due to improper food, sudden changes of diet, exhaustion from overwork, particularly if associated with long fasting, and to other circumstances, trivial in themselves, and quite insufficient as causes if uncombined with other disturbing influences. For example, a drink of cold water is often supposed to cause colic. Now water, no matter how abundantly it might be drank, as is witnessed in diabetes, does not cause colic; but if an animal be exhausted by a long journey, or bathed in profuse perspiration, cold water may then cause disturbance and abdominal pain.

Subcutaneous injections of chloride of barium, 12 to 15 grains, or of physostigma, 2 grains, are exceedingly serviceable in non-inflammatory constipation, but exceedingly dangerous where the condition is associated with signs of inflammation.

Colicky pains are also symptomatic of intestinal concretions, parasites, intussusception, mesenteric abscesses, and of diseases of other organs, such as the pleura, kidneys, liver, &c. On this account colic has been divided by some authors into true and false; the true including the colicky pains arising from all intestinal diseases involving structural change, and the false those from other causes.

Amongst other causes of colicky pains may be enumerated mesenteric abscesses, succeeding strangles or other suppurative disease, ulcers in the stomach, the irritation of numerous parasites, cancer, and chronic inflammation and thickening of the intestinal walls.

I have seen two cases of chronic induration of the duodenum and pylorus. The symptoms in both were as follows:—Capricious appetite, slight colicky pains succeeding in about two hours after a meal, a peculiar staring appearance of the eye, hanging back in the stall to the full length of the collar shank, a peculiar frightened look, irregularity of the heart's action, the

pulse sometimes slower, sometimes faster than natural, but always irregular; gradual emaciation and death. The *post mortem* appearances, a thickening of the submucous areolar tissue, which presents a whitish and fibrous character, and being united most intimately with the mucous and muscular coats, which were both pale, thickened, and contained much translucent material. The whole mass was firm, and resisted the knife.

*Symptoms.*—When colic is truly intestinal the symptoms are, sudden pain, pawing, kicking at the belly, looking round at the flanks, lying down, rolling, struggling in a variety of ways, or lying outstretched; then suddenly rising, shaking the body, and remaining for a short period free from pain. After a short interval, however, the symptoms return, sometimes in an aggravated, occasionally in a modified form, and this occurs again and again, until the animal is either relieved or dies from enteritis, pain, and exhaustion. During the paroxysms of pain the breathing is accelerated, sighing, or panting, the pulse is observed to rise in frequency, and to become more or less full and hard; during the intervals of ease it may fall to its normal condition. At the commencement of the attack there is generally a frequent evacuation of small quantities of fæces, which are sometimes hard, sometimes soft; the urine is passed in small quantities, or there are frequent but ineffectual attempts to micturate; and if an examination be made *per rectum*, the bladder will very often be found full and distended, the urine being retained by a firm contraction of its neck. In some instances, more especially if the animal has been fed on moist grasses, potatoes, or unripe corn, there is diarrhoea and escape of much foetid flatus.

The seat of the spasm is sometimes in the small, often in the large intestines; the symptoms during life, however, do not enable us to ascertain this with certainty. I have, however, observed that pawing with the fore feet, frequently looking round to the side, with very acute and sudden pains, point to the small intestines as being the seat of the spasm. In some instances there has been violent agony, a tendency to rear, and to kick savagely with one hind foot, when the disease has been confined to the small intestines, whilst a disposition to back or press the hind quarters against a wall or other solid object is almost a constant symptom of impaction of the colon.

*Flatulent colic*, whether occurring primarily or subsequent

to an attack of spasmodic, is a condition from which much more serious results are to be apprehended than the spasmodic.

Its causes are—weakness of digestion, but most commonly food which easily undergoes fermentation, such as raw potatoes, green clover, a mixture of maltcums and brewer's grains, wheat, and boiled food.

In this form of colic the expression of pain, though not so acute, is much more constant than in the spasmodic form; the abdomen is more or less tensely swollen, and resonant upon percussion; the pulse soon becomes rapid and feeble, the breathing difficult and mostly thoracic, the extremities become cold, there is more or less delirium, the animal reels to and fro; twitching of the muscles, retraction of the lips, and if relief be not afforded, death ensues either from asphyxia, blood poisoning from absorption of gases, or rupture of some portion of the intestines. When the animal lies down or rolls it is observed that it performs these acts much more carefully than when suffering from spasm.

Tympanites may arise independently of any cognizable extrinsic cause. Occurring during the progress of another disease, it is always to be looked upon as indicative of a very grave condition, that the animal powers are so exhausted as to be bordering upon dissolution, and becoming amenable to chemical laws. Tympanites also occurs in obstructions of the intestinal canal from calculi, tumours, or other mechanical causes, and generally indicates the approach of death.

*Treatment of Spasmodic Colic.*—Slight attacks are often permanently relieved by tincture of opium, with spirit of nitrous ether, administered as a draught with water, or with eight to ten ounces of linseed oil. If relief be not afforded, say in half an hour, and if the colic be not associated with inflammatory symptoms, an attempt must be made to remove the source of irritation by means of cathartics or aperients. The late Professor John Gamgee recommended as the sole treatment the administration of a ball containing from eight to ten drachms of aloes, according to the size of the horses, if fed upon dry food; but if the food were of a succulent nature smaller doses were to be given, with enemas of warm water. He condemned the use of anodynes and stimulants. Other practitioners prefer to give from twelve to twenty ounces of linseed oil, combined

with a stimulant such as carbonate of ammonia, spirit of nitrous ether, or with oil of turpentine; others prefer tincture of opium; others belladonna or tincture of aconite, Indian hemp, chloral hydrate, and other fancied anodyne or stimulating drugs. For my own part, I now prefer linseed oil with tincture of opium and spirit of nitrous ether, followed, if necessary, by sodium hyposulphite, one or two ounces dissolved in water, at intervals of three or four hours, with an occasional enema of warm water. *All cases of bowel affections should be examined per anum at the commencement of the attack*, as there is a possibility of detecting and removing a cause lodged in the rectum. The enema tube should consist of guttapercha piping at least four feet long, with a rounded nozzle, and introduced by gentle—not forcible—pressure as far as possible into the bowel. If the urinary bladder be distended, pressure from the rectum with the palm of the hand will often assist the act of urination; if this be insufficient, relief must be given by means of the catheter.

In many parts of the country colic is looked upon as a disorder of the urinary organs by non-professional people, and from the fact that the first sign of recovery is often the act of urination, countenance is given to this opinion. There is doubtless a spasmodic contraction of the constrictor vesicæ in the majority of colic cases; and relaxation of this spasm is a concomitant of that of the intestines; hence rapid recovery generally takes place after the act of micturition.

When the contents of the bowels are found by rectal examination to be hard, an attempt should be made by manipulation or kneading with the hand in the rectum to break down the impacting masses, and thus assist the bowels to resume the peristaltic action.

In conclusion, it may be stated that if the attack be a very slight one, a single dose of the opiate will often give permanent relief. There is one thing that I should warn the practitioner about, and that is, no certain dependence can be placed upon this, and that in all cases where the animal cannot be watched for some time afterwards the aperient is not to be omitted; and upon no account is a horse attacked late at night, and thus relieved, to be left without the aperient being administered, for it has often happened that when all are asleep in bed the pains have returned, and the horse in the morning found dying or dead.

*Treatment of Flatulent Colic.*—In addition to the administration of an aperient for the removal of the cause, it becomes essentially necessary that the symptoms of urgency be promptly relieved. Carbolic acid or the hyposulphite of soda to some extent averts fermentation and the further formation of gases, and thus prove very useful. If, however, the tympanites be severe, or seem to increase under the prescribed treatment, the colon should be immediately punctured with a Toop trochar and cannula, to enable the gases to escape, and thus give immediate and permanent relief. The method of procedure is as follows :—Select the most prominent part of the swelling, which is generally about midway between the last rib and anterior spine of the ilium, upon the off—right—side ; asepticise the spot, then make an incision with a small bistoury, and introduce the point of the trochar rather obliquely upwards, in order to allow the escape by drainage of any fluid which may be afterwards exuded, and thus prevent the formation of an abscess ; when the bowel is punctured, the stilette is withdrawn, when the gas will be freely expelled ; and in order to prevent further fermentation it will be necessary to inject, by means of a Toop syringe, through the cannula, about three-quarters of an ounce of pure carbolic acid, dissolved in twenty ounces of warm water, into the intestine. I feel confident that we have saved many horses in the College practice since adopting this treatment.

If the tympany is not entirely removed by the escape of the gases, the operation may be performed on the opposite flank with great benefit. There is no danger in the operation : in one case it was performed thirteen times, giving relief of several hours' duration after each puncture, in a case of tympany associated with a large calculus in the colon. The horse lived several days, and on making an examination of the seats of puncture only three or four small reddish spots were seen, with no surrounding inflammation.

#### IMPACTION OF THE COLON.

As already stated, animals over-abundantly fed, or kept upon food containing much woody fibre, are liable to suffer from the accumulation of such matters in the colon and cæcum. The diagnostic characters of this, in addition to symptoms of general abdominal pain, are a tendency to push backwards, to press the

tail against any solid object, to resist by violent straining the introduction of the hand and enemas into the rectum. In some cases the hard and impacted mass may be felt by the hand introduced into the rectum, and there is also some enlargement or distension of the abdomen. The propriety of administering cathartics by the mouth for the relief of this condition is a question of very great importance, for it has often happened that rupture of the colon has occurred in the course of some hours—generally about twenty—after the administration of an aloetic ball, the condition of the intestinal canal, when examined *post mortem*, having been found as follows:—The stomach and small intestines more or less emptied of alimentary matters, or containing large quantities of fluid; the large intestines distended with a mass of more or less hardened material, or partly hard and partly soft; the mucous membrane sometimes highly congested, sometimes pallid, but ruptured, and the contents more or less escaped, as in ruptured stomach, into the peritoneal cavity. I have repeatedly witnessed this, and the question has arisen in my mind whether the rupture has not been induced by the contents of the small intestines having been forced into the already over-burdened large ones by the action of the purgative. Concluding that this was the case, I have injected aloetic solutions into the rectum, and endeavoured to excite the commencement of purgation in the large intestines. In some instances this has succeeded, whilst in others it has failed; the failure has been due to the expulsion of the medicine by the animal. I have found it the best method to dissolve two ounces of aloes in a pint of hot water, and to administer it with a syringe having a long flexible tube, at a temperature of about 90° F. If this be retained for an hour or two, it generally has some effect, but if immediately expelled, it ought to be repeated. I am well aware that writers on materia medica will say that the specific action of aloes is limited to the large intestines and rectum, no matter how it gains entrance into the economy, and that this result will be obtained by its administration by the mouth. My experience leads me to the conclusion that, although it may irritate the posterior bowel during its exit from the body, its primary effect is to stimulate the peristaltic action and glandular secretion of that part of the intestines with which it first comes in contact.

Rupture of the colon from impaction, or when resulting from

degeneration of its muscular coat, is manifested by symptoms similar to those of rupture of the stomach. If occurring from impaction, it will be found that the passage of enemas, which had previously been difficult, owing to the violent straining or resistance of the animal, becomes suddenly easy; the hand introduced into the rectum meets with little or no resistance, the intestine itself being dilated and paralyzed.

#### DISEASES OF THE RECTUM.

The condition of degeneration observable in the stomach and colon is sometimes met with in the rectum, causing a gradually decreasing contractile power of the bowel, in consequence of which the faecal matters accumulate to an inordinate extent, and are only expelled by violent straining and contraction of the abdominal muscles. In such instances, it is observed that the faeces of the horse, which in its normal condition is divided into globular pellets, is discharged from the body as a huge mass, resembling a large German sausage, and as thick as a man's leg. I have observed this condition, which may be denominated paralysis of the rectum, as a result of injury of the spine or sacro-lumbar nerves, and independently of injury in old horses habitually kept upon coarse, indigestible food. When resulting from injury, it has sometimes disappeared, either spontaneously, as the lesion which caused it became repaired, or by the assistance of nervine tonics, more particularly nux vomica, and blisters to the sacro-lumbar region. In many instances, it has been necessary to empty the bowel by the hand four or five times daily, and to administer enemas to prevent pain and colicky symptoms. Rupture of the rectum has sometimes occurred from this cause.

*Rupture of the rectum* has also occurred within my experience from the formation of an aneurism of the hæmorrhoidal arteries, the pressure of the aneurismal tumour having caused atrophy, not only of the muscular and peritoneal, but also of the mucous coat of the intestine, and rupture of these being caused by some sudden effort; hæmorrhage has occurred both into the intestinal canal and peritoneal cavity. Rupture has also been witnessed as a result of a tumour, and as an accident during parturition.

Accidental rupture of the rectum, posterior to its peritoneal



lining, is not necessarily fatal; but if anterior to the termination of the peritoneum, the escape of fæcal matters and of blood into the peritoneal cavity will cause death. In one case, where the rectum was pierced by the foot of the foal during parturition, causing a recto-vesical fistula, I was enabled to bring the lips of the wound together by metallic suture, and induce their ultimate union, the mare eventually doing well.

*Inversion of the rectum* is caused by violent straining during parturition, or attempts to expel fæcal matters; as a result of paralysis; in the coma and spinal paralysis witnessed in parturient apoplexy in the cow; and in pigs it is a common occurrence owing to constipation. In dogs it results from the impaction of bones or other hard bodies in the bowel, causing violent straining. Its reduction in all animals must be preceded by thoroughly emptying the bowel of all sources of irritation. When this is effected, the bowel, thoroughly washed and oiled, is to be carefully returned into its proper position, and retained there by a suture across the anus, or a rope or truss properly adjusted (West's clamp), and the sense of pain and irritation soothed by opium. For several days the bowel is to be carefully emptied by the hand, or enemas, and the animal fed upon a restricted diet. Purgatives are to be avoided; the bowel may, however, be lubricated with oleaginous enemas.

In recurring inversion, which commonly happens in the pig, excision of the protruded intestine may be successfully performed; and in all cases where it is found impossible to return the bowel, the engorged mucous membrane is to be carefully dissected from the subadjacent structures, and a reduction thus effected. It now and then happens that the sphincter of the anus closes firmly, forming a constricted neck, preventing the return of blood to such an extent that the protruded intestine speedily becomes gangrenous. If the sufferings of the animal be not very great, it is generally advisable to allow a short time to elapse before removing the sphacelated mass, in order that new adhesions may form; but if the animal suffers severely, it is advisable to insert sutures for the purpose of causing union between the gut and anal opening, and at once remove the whole protruded mass with a sharp bistoury, the subsequent pain and straining being alleviated by a full dose of opium. When several feet of the intestine are protruded, as sometimes happens after parturition, it becomes an impossibility to return

it. The intestine is generally lacerated from being trodden upon, and humanity renders it necessary that the animal should be put out of its sufferings.

#### HÆMORRHOIDS OR PILES.

With the exception of the dog, the domestic animals rarely suffer from piles, which consist at first of congestion of the mucous membrane at the verge of the anus, and subsequently of dilatation of the hæmorrhoidal veins, constituting small tumours. These sometimes protrude outside the anus, and bleed frequently. The diagnostic symptoms of piles are switching of the tail, and a tendency to rub it against the wall; pain during the act of defecation, and the fæces being tinged with blood. The dog sits on its haunches and pulls itself along in that position. The examination of the anus will reveal the presence of vascular tumours, and of much congestion and swelling of the mucous membrane and skin.

*Treatment.*—The cause of hæmorrhoids depends upon some obstruction to the portal circulation, constipation, and frequently on retention of hardened fæces in the rectum; the treatment must therefore be directed to the removal of these by manipulation, gentle laxatives, and a restricted diet. The uneasiness may be modified by fomentations, enemas, and the application of some mild astringent. Astringent ointments are also useful, more particularly an ointment consisting of equal parts of oak galls and hog's lard; the benzoate of zinc ointment is also a useful application.

Preparations of adrenalin, especially the ointment, if properly made, are particularly useful in the case of small dogs. It being non-irritating and easy of application, the effect is almost instantaneous in those objectionable cases of pet dogs who, by the irritation produced, perform gymnastic feats in their endeavour to remove the cause of irritation.

#### IMPERFORATE ANUS.

A congenital malformation met with in all the domesticated animals, particularly in the pig. It gives rise to symptoms of colic, and generally proves fatal shortly after birth. In some

instances the anal opening is well formed, but the rectum at a short distance from it forms a *cul de sac*, which is not continuous with the alimentary canal. In other instances the anal opening is permanently closed by skin, the bowel terminating in a pouch underneath it. When this condition is met with, relief is possible by puncturing the intestine, thus forming an artificial anus.

## CHAPTER LXIX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

#### INFLAMMATORY DISEASES OF THE INTESTINES— ENTERITIS.

ENTERITIS, or inflammation of the bowels, may safely be stated to be the most rapidly fatal inflammatory disease to which the horse is liable, destroying life in the course of a few hours. Indeed, it is very doubtful whether the disease recognised as enteritis by veterinarians is a true inflammation at all, as its course and progress, rapid termination, and *post mortem* appearances tend to confirm the opinion that it partakes more of the nature of apoplexy than of inflammation. It is very true that impaction, constipation, intussusception, the presence of calculi, or the action of irritant poisons, may cause great congestion and inflammation of the intestinal membranes; but the disease to be described originates *sui generis*, and very often without the occurrence of an immediate and recognisable cause of direct irritation. This fact has led some practitioners of very great experience, amongst whom may be mentioned the late Mr. Lawson of Manchester, to arrive at the conclusion that enteritis never originates in colic, spasmodic or flatulent. Many writers assert that enteritis is situated in the small intestines, more particularly in the ileum and jejunum. My experience, however, leads me to the conclusion that it is more commonly situated in the cæcum and colon than in any other part of the intestinal canal, but no portion of the tube is exempt. The only recognisable causes are, over-fatigue, cold

from exposure, or from washing with very cold water whilst the animal is heated, and thereafter inadequately clothed.

#### PATHOLOGY.

Various writers state that the inflammation is situated in the muscular coat. This is evidently a mistake, as the congestion is of the greatest severity in the mucous membrane, the redness of which is of a deep venous colour, approaching to blackness, in patches of various extent, and associated in many cases with extravasation of blood into the canal. Of course, when such extreme congestion of the mucous membrane exists, all the coats are more or less implicated, but the primary and gravest condition is limited to the mucous membrane.

The submucous tissue is generally much thickened; there is loss of cohesion, the mucous membrane being easily stripped from its attachments; some effusion of serum into the intestinal canal, causing purging in cattle and dogs; but in the horse purging seldom or never occurs, though the contents of the bowels may be found fluid after death. This seeming constipation results from paralysis or loss of function, and is marked by complete retention of the fæcal matters. In some rare instances enteritis may terminate favourably in the horse; but in the great majority of cases mortification results, or the animal dies from the debilitating effects of hæmorrhage into the intestinal canal, or from prostration of the nervous system induced by pain. Gangrene may result in eight or ten hours, the animal rapidly succumbing. In some instances, however, death may not result for several days. In one instance a horse lived for a period of five days, the bowel being found sphacelated, the process of ulceration having commenced at the edges of the gangrenous patch. It is very rarely that ulceration occurs in the horse, but it may be witnessed both in cattle and dogs.

#### SYMPTOMS.

The first noticeable signs are those of abdominal pain; generally, however, they are preceded by some degree of constitutional disturbance, rigors, accelerated breathing, repeated evacuations of small quantities of fæces, and general depression;

the mucous membranes soon become deeply congested, the mouth dry, the tongue contracted, and now and then of a brownish colour, the appetite of course being lost; the pulse is hard, wiry, and quick; the belly is tender upon pressure; the abdominal muscles more or less contracted; and if tympanites be absent the belly may seem tucked up and smaller than natural. By-and-by the symptoms of dulness and depression give place to those of excitement and pain; the horse stamps the ground with the feet, strikes at the belly, lies down, but much more carefully than in spasmodic colic, or makes feints to do so; it may roll upon its back; turns its eyes anxiously towards the flanks, pants, blows, and sweats with pain. There are no sharp paroxysms of pain with intervals of ease as in colic, but the pain is constant, distressful, and agonizing; in some cases so much so, that the sufferer seems afraid to express it, except by a most anxious expression, which is a characteristic and diagnostic symptom. Now and then the animal will stand persistently with the head in a corner, and paw the ground for hours together with one or both fore feet alternately. The pulse is hard, wiry, and quick, often ranging from 80 to 120 beats per minute; and as the disease advances, it becomes thready and imperceptible; the animal sighs, or even groans with pain; the perspiration runs off the body; the skin is never dry, at one time hot, at another cold; the countenance becomes haggard, the eyes expressive of delirium, pupils dilated. The horse may throw itself about in a most dangerous manner, or walk round its box incessantly; then it will stand, balance itself, its legs give way, when it may fall and die after a few convulsive struggles, or suddenly all symptoms of pain may subside; it will then stand quiet, and even drink or endeavour to feed; its breathing becoming more or less tranquillized; but the haggard expression of the face still remains; the pulse continues thready and imperceptible; cold sweats bedew the body; the belly becomes tympanitic; gangrene has now set in; it trembles incessantly; the legs and ears are deathly cold; the mouth cold; the breath cold, and even foetid; the lips drop pendulous; the eyes become more amaurotic, and after a varying interval death closes the scene, the bowels remaining inactive to the last. If, however, in three or four hours from the commencement of attack there be some abatement of the symptoms; if the surface of the body

become dry, if there be a passage of flatus or fæces, the pulse becoming fuller and softer, and the characteristic anxiety leave the expression, a favourable termination may be anticipated. This, however, is rarely the case.

#### TREATMENT.

Concluding that the constipation which is so prominent a symptom in enteritis is due to obstruction from alimentary matters, veterinarians generally administer powerful cathartics. It has, however, been shown quite conclusively that it is always dangerous to propel fæcal matters through an inflamed portion of bowel; and that in most cases the effort is useless, so far as exciting peristaltic action in the inflamed portion is concerned. Purgatives, however, stimulate and excite the muscular and excretory action of the healthy intestines anterior to the inflamed part; the result of this is, that the inflamed part becomes more and more distended, inflammation is increased, the blood-vessels become ruptured; it becomes softened, and its vitality is ultimately destroyed. It is therefore a fact that the inflamed part, the function of which is lost, consequent upon the inflammation, affords an impediment, and that purgatives, as a rule, have no true purgative effect—that is to say, they do not cause the discharge of fæcal matters by the anus.

Two great principles are therefore recognised, namely—first, to relieve pain, and, second, to arrest as far as possible all movement of the intestines; and for these purposes opium is to be administered in large doses. For the horse, one, two, or even four drachms of the powder may be administered, succeeded by smaller quantities at short intervals, or by the subcutaneous injection of morphia (the solution of the meconate of morphia being recommended. The first subcutaneous injection should contain, in addition to 5 to 8 grains of morphia, a half grain of atropia; but the atropia should not be repeated for at least twenty-four hours, whilst the morphia may be administered every few hours, according to the severity of the symptoms. In addition, hot fomentations to the abdomen are useful; and they should be continuously applied for at least an hour at a time. Enemas of warm water may also be gently administered: they are not, however, to be repeated too often, and if at any time they increase the pain, they should be discontinued.

If, after the abatement of the active symptoms, the bowels remain torpid (as they generally do), the practitioner is by no means to attempt the removal of this torpidity by the administration of cathartics or aperients of any kind; for if the animal is to recover, it must be dependent upon the restoration of function and tone to the inflamed bowel; and it is well known that for this end perfect quietude of the inflamed part must be maintained.

The advisability of bleeding in enteritis will depend entirely upon the condition of the animal. If the pulse be moderately full, if depression be absent, an abstraction of blood commensurate with the strength of the vital powers will be followed generally by abatement of the symptoms, and if performed early is beneficial. Should the appetite return, great care must be taken that the food be of the simplest and of the most easily digestible kind, such as scalded bran and boiled linseed given in moderate quantities; the eating of dry food being prevented by a muzzle. Care must also be taken, when the functions of the bowels are restored, that no undue accumulation of fæcal matters be allowed to remain in the rectum. The question of administering stimulants during the acute stage will also greatly depend upon the condition of the animal; they usually do more harm than good, but if tympanites be present, one or two doses may be tried. If they give relief they may be continued, but if, on the contrary, they aggravate the pain or seem to have no effect, they are to be discontinued. It must not be lost sight of that antiseptics, such as carbolic acid or hyphosulphite of soda, in proper doses are important factors in counteracting putrefaction of the contents of inflamed bowels, and the rapid gangrene of the inflamed part, as well as a septic condition of the blood. If, however, they fail to reduce the tympany, relief by puncture must be immediately given (see *Treatment of Flatulent Colic*).



## CHAPTER LXX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

#### INTUSSUSCEPTION AND VOLVULUS.

INTUSSUSCEPTION (from *intus*, within, and *suscipio*, to receive), and volvulus (from *volvo*, to roll up), a twisted bowel. Under these names various forms of entanglement of the intestines, giving rise to abdominal pain, enteritis, and death, have been described.

#### INTUSSUSCEPTION.

By *intussusception* is meant the prolapse or slipping of a portion of intestine into the cavity of that immediately posterior to it. In consequence of this the natural course of the intestines is interrupted by a kind of knot, consisting of three successive portions of bowel; the immediate effect of which is obstruction to the passage of the intestinal contents, and to the return of blood from the imprisoned portions of intestine involved, along with which some portion of the mesentery must be included.

Both the small and large intestines are subject to it in all the domesticated animals. When, as is generally the case, it occurs in the large intestine, the cæcum caput coli is that usually involved, and instances have occurred, both in horses and cattle, of recovery after sloughing of the invaginated cæcum. Within my own experience, a cow treated by me, after seven days of obstinate obstruction of the bowels, expelled the gangrenous portion, measuring several inches in diameter, and the animal recovered.

Mr. Aitken, V.S., Dalkeith, has a case on record of a cow which recovered after five days of illness, the gangrenous portion being expelled with the fæces. Before recovery from intussusception can occur, it is necessary that the opposed peritoneal surfaces become adherent, and the imprisoned portion separated by ulceration.

Invagination of the small intestines, owing to the severity of the inflammation, is necessarily fatal.

*Symptoms.*—In the cow already mentioned, the symptoms were those of enteritis and obstinate constipation. The treatment consisted in the administration of opium. In Mr. Aitken's case drastic cathartics were administered from the commencement. The symptoms in the horse, as related by the late Mr. John Field, are as follows:—"Pain; restlessness, in some cases approaching to madness, unrestrainable; wandering about; rolling on the back; sweating, in some cases profuse; crouching; sitting on the hind quarters, almost diagnostic; anxious countenance; frequent feeble pulse; belly at first of natural size, subsequently fuller, in some cases distended, dependent upon the locality of the intussusception; membranes, in advanced stage, turgid, injected; mouth moist and clean, or furred and offensive; respiration accelerated; continued restlessness; rearing with fore-legs into manger, and standing upon that *point d'appui*, looking back from side to side; extremities cold; pain absent, tranquil; sighing or snorting; death. The sighing may exist in some cases and not in others, and in some retching and vomiting."

I am, however, of opinion that there is no diagnostic symptom of volvulus, intussusception, calculus, or strangulation of the intestines in the horse; that the above symptoms are common to all, characteristic of none. In the dog and pig, however, vomiting of stercorous matters is generally witnessed; but even in these animals this symptom may be induced by any cause of obstruction. Stones accidentally swallowed, pieces of bone arrested in the small intestines, are frequent sources of obstruction in the dog.

*Treatment.*—It has been proposed to cut down upon and mechanically remove the source of mischief. I think, however, that the operation would be as bad as the lesion. If intussusception be due to contraction of one portion of intestine, and

the slipping in of that contracted portion into the healthy portion behind, remedies calculated to relieve spasm may prevent its occurrence, but cannot overcome it when once established.

#### VOLVULUS, OR TWISTED BOWEL,

May occur in either the small or large intestines, and, like strangulation of the intestines by pedunculated tumours, or from their entrance into the inguinal canal or the foramen of



FIG. 81.—Twist of the bowel.

Winslow, are but rarely witnessed, even in the horse, and their exact diagnosis is attended with difficulty. It is true that some practitioners assert that when a horse resists the introduction of the hand into the rectum by straining, that it is indicative of volvulus of the colon; but if, on the contrary, the rectum be found passively distended—hollow—that it indicates impaction and paralytic loss of function of that bowel. I have seen many cases in my time, and can safely say that these diagnoses are unsupported by extended observation. Many theories have been advanced to explain the etiology of volvulus. A case published in the *Veterinary Journal*, January 1897, by Mr.

Locke, M.R.C.V.S., Manchester, was likely induced by several falls while a high-spirited horse was taxed beyond his strength in trying to pull a heavy load out of a deep place. Rolling whilst suffering from colic has been perhaps rightly blamed, and aneurisms of the mesenteric artery, by causing anæmia of a portion of the bowel, has also been ascribed as a cause.

The diagnosis is extremely difficult. There is continuous pain, —mild perhaps at first when the large colon is twisted, severe from the first when the small bowel is involved; borborygma is absent, and no fæces passed after the very earliest stage; on rectal examination there may be a difficulty, if the backward pressure is great, in introducing the hand into the rectum.

Jelkman states that there is no difficulty in diagnosing volvulus per rectum, and even of effacing it by manipulating through the bowel. Copious enemata of oil and warm water, administered through a long tube, and the administration of anodynes—morphia or chloral hydrate—with attempts to reduce the volvulus by manipulation, by rolling the animal, and, as a last resort, laparotomy might be performed. As a rule, however, the progress of the congestive change is so rapid that there is little chance of saving life. Anæsthesia will render reduction a much easier matter, as well as give the animal at least temporary relief.

## CHAPTER LXXI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

#### INTESTINAL CONCRETIONS.

THE common seat of the various concretions is found to be in the large intestines, where they sometimes attain a large size, and as much as twenty-five pounds or more in weight. Occasionally, but very rarely, they have been found in the stomach weighing four or five pounds.

They were studied by the late Professor Morton, and classified under three heads, namely, 1st. Phosphatic calculi; 2d. Oat-hair calculi; and 3d. Mixed calculi.

1st. *Phosphatic Calculi.*—These are hard, smooth, and polished on their external surface, bearing much resemblance to a common pebble. If a section be made of a phosphatic calculus, it will be found to consist of concentric layers arranged around a nucleus, generally consisting of a piece of iron or stone. If several of them, or any other form of calculus, be formed together, they are generally flattened upon their sides, or concave on one side and convex on the other, the convexity of one stone fitting into the concavity of another, and so on. This form of calculus is now much more rarely met with than formerly. This arises from the circumstance that the food of animals is much better prepared and cleaner than it used to be, and containing, therefore, fewer accidental materials or foreign bodies, which formed the nuclei.

Their chemical composition, according to Girardin, is as follows:—

Ammonio-phosphate of magnesia,	.	.	48·00
Phosphate of lime,	.	.	19·00
Water,	.	.	14·00
Animal matter,	.	.	0·80
Soluble salts, &c.	.	.	6·60
Extractive matters,	.	.	4·00
Fatty matters,	.	.	7·00
Loss,	.	.	0·60
			<hr/>
			100·00
			<hr/>

From the above analysis it would be seen that the calculi contained some animal matters; in fact the earthy materials are glued together by mucus, &c.

Their occurrence is accounted for as follows by Professor Morton:—"In the cereal plants, certain of the phosphates are met with, and in considerable quantities. It is then to this food that we are to look for their origin, coupled with a morbid state of the digestive functions, by which it does not undergo the necessary change, probably from the gastric juice not being sufficiently powerful to dissolve these phosphates. A foreign body being accidentally taken into the stomach, which may be a nail, or anything else, as a piece of stone, serves as a common centre, the phosphates arrange themselves in their turn, and in doing so they blunt that which by its sharpness would wound the lining membrane of the organ. If from its magnitude the calculus is unable to pass through the pylorus, the stomach becomes its residence. They will give rise to colicky pains, depravity of appetite, perhaps to wind-sucking or crib-biting, and in the end will invariably, by accumulation destroy the life of the horse.

2d. *Oat-hair calculi*, generally found in the cæcum or colon, consist almost entirely of the beard of oats, barley, or other grain. They sometimes attain a large size, but are light in comparison with the first-named. They, however, assume the same shape, and are occasionally mixed with phosphatic salts.

3d. *The mixed calculi* are composed of phosphatic salts, faecal matters, oat-hair, or any indigestible matter which may accumulate in the intestine.

In addition to these, food may adhere to the mucous membrane, constituting what is termed a stercoral concretion, con-

sisting very often of straw-knots, &c., firmly glued together. This will sometimes be found perforated through its centre, so as to admit the passage of fæces.

The presence of these concretions at some time or another constitutes a fatal obstruction. Many symptoms are laid down as being diagnostic of calculus, such as sitting on the haunches, lying upon the back, &c. I am not, however, aware that there is any diagnostic sign, beyond those of violent abdominal pain and obstruction, by which they may be discovered during life; and they can only be guessed at by recurrent attacks of colic and constipation. In one case which fell under my notice there were no symptoms of abdominal pain, but merely constipation, which nothing could overcome. For two days after the animal had been observed to be off its food the pulse remained natural; on the third day, however, the abdomen became tympanitic, the pulse rose rapidly, and the animal died; and the *post mortem* revealed the presence of a phosphatic calculus, about ten pounds in weight, firmly lodged in the single colon. In one case examination *per rectum* enabled me to feel and remove a calculus from the rectum, the animal being immediately relieved. This shows the necessity of such an examination in all cases of abdominal pain.

Cases are recorded of polypi or other tumours in the stomach and in the intestines. Gastric polypi finally plug up the pyloric orifice, and give rise to fatal distension.

To prove the fallacy of forcing the action of the bowels in cases of mere constipation, and to show how long a horse may live without passing any fæces, I may mention that I had a horse under my care that had no action of the bowels whatever for twenty-five days, yet it made a good recovery. It was noticed that after a purgative had been administered the pulse became greatly accelerated, mucous membranes injected, and that it manifested signs of pain, which continued for several hours, in fact until the medicine was excreted by other organs. Except when suffering from the effects of medicine it ate a moderate quantity of food. Another case lived eighteen days, passing no fæces, and showing no pain except when irritated by purgatives; towards the end of that day it showed pain, and died from calculus in the single colon. Upon this point the following letter from a late student, Mr. Martin, now in London, is instructive:—

“ LONDON, 6th June.

“ MY DEAR SIR—In reply to your note you kindly sent me of 29th ult. in reference to the case of stoppage of the bowels of a horse I had under my care, I have now to inform you that he died on Monday evening, May 26, it being the thirtieth day that no fæces had passed from him. Within six hours of his death he started blowing, suffering a deal of pain, abdomen distended very much. Next morning, on *post mortem* examination, I found the bowels gorged, and a calculus firmly fixed in the single colon, weighing  $2\frac{1}{2}$  pounds, 5 inches in diameter. All the other parts were perfectly healthy. It appeared to me that the bowels becoming so full, caused him to die sooner than he would have done had less solid food been given him, although the result would have been the same.”



## CHAPTER LXXII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

##### DIARRHŒA.

THIS term is applied to all cases of simple purging in which the fæces are loose, liquid, and frequently discharged without any coexistent inflammation. Diarrhœa may be a spontaneous effort to discharge from the intestines something which is obnoxious to them or to the system generally. It is also induced by a variety of causes in all animals, such as indigestible food; sudden changes of diet, particularly from a dry to a moist one; medicinal substances; parasites; derangement of the liver; or copious draughts of water when the animal is heated.

Some animals are particularly prone to diarrhœa from trivial causes; narrow-loined, flat-sided, and loosely coupled horses—that is to say, horses in which the space between the ilium and last rib is large—and those which are of a nervous temperament, are apt to purge without apparent cause. They are vulgarly called “washy” horses. Such horses will start upon a journey in the best of apparent health, but before they have proceeded any great distance, will commence to purge more or less freely. They are hard to keep in condition, and require the best of food. If, however, put to slow work they will sometimes do well enough.

##### SYMPTOMS.

The symptoms of diarrhœa are purging, the faecal matters being semi-fluid, of a dirty brown colour, without offensive odour, or clay-coloured and fœtid. If the condition continues long, the animal loses flesh, the appetite fails, and in some cases ascites, or even farcy and glanders, may supervene.

## TREATMENT.

When the purging arises from the presence of some offending matter in the intestinal canal, its expulsion must be aided—and this applies to all our patients—by a moderate dose of castor or linseed oil, and the diet must be changed. Purging in the dog is often induced by a too exclusively farinaceous diet, and it is impossible to arrest it without an entire change of food. If the purging arises from no apparent cause, or if the bowels do not regain their normal condition after the action of the aperient has subsided, it will be necessary to administer calmatives, such as opium or mild astringents, such as chalk; proceeding, however, very cautiously, as too sudden a check may induce complications of a grave character. Boiled starch or flour gruel may be allowed the animal to drink; the food must be of the sweetest and best kind, and given in moderate quantities.

If there be much fœtor the sulphite or hyposulphite of soda may be very advantageously given dissolved in the drink, or mixed with the food. If the animal be depressed and weak, moderate and repeated doses of the spirits of nitrous ether will afford relief, and promote the restoration of the bowels to their natural condition. Should this treatment prove futile, the more powerful astringents, such as catechu or kino, or, what has proved more successful with me, the oil of turpentine and opium beaten up with eggs, are to be administered. Some practitioners recommend the mineral acids, such as the aromatic sulphuric acid, these remedies having a tonic as well as an astringent action upon the bowels. Warm clothing and perfect quietude are necessary.

The fact of the possibility of the presence of parasites in the intestinal tract must never be lost sight of in all cases of digestive disorder, and especially so in the cases of the smaller domesticated animals. A diagnosis can usually be made by microscopical examination of the fæces for eggs (*vide* Parasites). Treatment in accordance should be adopted with a view to expelling the troublesome guests. In such treatment thymol may be tried. Its efficacy is increased by frequent small doses, rather than large and infrequent administrations. It is especially useful in nematode affections.

## CHAPTER LXXIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

##### SUPERPURGATION.

ALTHOUGH a form of diarrhoea, from its gravity, and the important legal questions it may give rise to, superpurgation deserves a separate consideration. It is well described by the late Mr. Haycock. He says—"Superpurgation, or over-excitement of the intestines from the undue action of purgative medicine, is a condition of the bowels which the veterinary surgeon is frequently called upon to treat. The symptoms associated with the action of a purgative upon the bowels are quickened rate of the pulse, attended with a partial but temporary loss of its force; the breathing also is a little hurried. This is the most conspicuous when purging is about to commence, and the animal is nauseated. If the purging, however, does not go on to an undue extent, these symptoms soon subside, the pulse becomes normal, and the nausea succeeded by a desire on the part of the animal for food; but should the animal be of weak constitution, or be taken out of the stable and ridden or driven a considerable distance while the purging continues, or taken out too soon after what is called the 'setting' of the physic, or if the purgative dose be too powerful in the first instance, or if two or more of these circumstances or causes act in association, the purging will in all probability become excessive, and the life of the animal may be placed in danger. Purging may continue for a long time; but so long as the patient is kept quiet in the stable, so long as the appetite remains good, and the pulse maintains its regular, slow, and round beat, little or no danger need be apprehended;

but if the appetite fail, if the pulse becomes thready, and the patient weak, it behoves the owner to act with promptness." "The following symptoms," says that excellent observer John Field, "indicate the violent and too long-continued action of purgatives, and invariably portend a fatal termination:—Staring glassy eyes; frequent, indistinct, feeble, and sometimes thread-like pulse; purging offensive matters, with or without distension of abdomen, or distended abdomen without evacuations; offensive mouth; tongue pallid or whitish with fur, and pasty; smell quite peculiar; respiration tranquil, but it becomes laborious when the belly becomes enormously distended; extremities warm; the horse usually stands still, sometimes paws or wanders about, and but rarely lies down."

Superpurgation does not always depend upon the strength of the dose. In some instances as little as four drachms of aloes have been succeeded by fatal consequences. Again, horses in an obese condition, and those suffering from slight colds, are easily acted upon by purgative medicines, and are apt to sink from superpurgation. A full dose of aloes, from six to eight drachms, operating quickly, is seldom succeeded in healthy animals by any evil consequences; the same quantity, however, if divided into two or more doses, has a much more depressing effect, and is apt to be followed by serious consequences. In the first instance, the quantity, by its strength, insures its own expulsion; whilst in the second, the aloes is absorbed into the circulation, excites a toxic effect upon the system generally, and reduces the horse to such a state of debility that it succumbs to the purgative influence. The explanation of the tendency to superpurgation in the horse is to be found in the fact that its bowels are extremely vascular in comparison with those of other animals, and that the effect of the purgative acting upon so vascular a surface is grave and serious. I have already pointed out that many young horses, when first brought into the stable, are rendered susceptible to various diseases by the debilitating influences of indiscriminate purging, and that such a method of treatment is uncalled for and irrational. In addition to the symptoms described by Messrs. Haycock and Field, I have observed that those of laminitis are induced by purgatives, and that when they occur they indicate a condition of great gravity.

The *post mortem* appearances are those of congestion of the

intestinal mucous membrane generally, concentrated in many cases in that of the *cæcum caput coli*; a thick, tarry appearance of the blood, and extreme blackness, congestion, or apoplexy of the lungs, the blood being, as it were, deprived of its watery elements, altered in its composition, rendered too viscid to circulate through the pulmonary capillaries, and so altered chemically as to be rendered unfit for perfect oxidation.

In order to prevent the occurrence of superpurgation after the administration of an aloetic, or, more particularly, a mercurial and aloetic purgative, it is necessary that the practitioner should order the animal to be fed on an easily digestible diet, such as warm bran mash; that the quantity of water should be restricted, and that the chill be taken off it, for nothing is so apt to induce inordinate intestinal action as large quantities of cold water whilst the animal is in physic. It is also necessary that no green food, roots, or other articles of diet, containing much water, and laxative in themselves, should be allowed at this period. If a purgative does not seem to take effect in from twenty to twenty-four hours after its administration, moderate walking exercise is to be prescribed, for it is a fact that the longer a purgative is retained in the body, the greater the danger from its superaction; if, however, purging has actually commenced, exercise, by increasing it, is apt to cause harm. It is therefore necessary to keep the horse quiet until the physic has "set."

#### TREATMENT.

So long as the horse remains moderately lively, the pulse but slightly accelerated, the countenance natural, and so long as some appetite remains, it is unnecessary to take any active measures to restrain the purging, which is the natural and physiological response of the intestines to the action of the cathartic; it is therefore irrational and dangerous to check it, and all that is requisite is to allow the horse to partake of demulcent drinks, such as thin flour gruel, if it will do so spontaneously, at the same time keeping it perfectly still, warmly clad, and taking care that it does not drink too freely of anything whatever. Should there be any colicky pains, moderate doses of opium are to be administered. Care, however, must be taken that the purging be not checked even by these means too

suddenly; as the consequence of this would be congestion of the intestinal mucous membrane, denoted by tympanites, great prostration, cessation of the purging, and the other symptoms described by Mr. Field, finally terminating in death.

If the purgation continue, an endeavour should be made to overcome it gradually but not too quickly. For this purpose I know of nothing better than tincture of opium, chalk, and flour gruel. Two ounces of the tincture are to be given, with the same quantity of prepared chalk, mixed with a quart of flour gruel, every three or four hours until the purging is checked, hot applications being in the meanwhile applied to the abdomen, great care being taken that the animal has no access to cold water or other fluid, as its thirst is great and it is apt to drink inordinately; but it is essential that it should have small quantities of flour gruel or other emollient drink, not only to allay the painful and feverish thirst, but to keep the blood in a proper fluid condition to circulate through the minute pulmonary capillaries. If the prostration be very great, stimulants, as wine or brandy, are to be tried; if they seem to act beneficially, they are to be continued, but not otherwise.

As a medico-legal question, the occurrence of superpurgation after the administration of a simple and moderate cathartic by the veterinarian is one of great importance. In no case where due caution has been taken, where the dose has not been more than the necessities of the case required, and where the above mode of after treatment has been enjoined, should the veterinarian be held responsible.

Superpurgation is typical of inflammatory diarrhœa, no matter what its cause may be, and as the symptoms and treatment are identical, there is no necessity for further description. It may, however, be mentioned that a form of diarrhœa, even more fatal in its consequences, accompanied by a greater degree of prostration, and terminating fatally in a shorter period of time than that induced by medicinal cathartics, is brought about by an over-abundant feeding upon raw potatoes. Again, the same condition is induced by the ingestion of food containing sand, small pebbles, or other materials incapable of being digested. There is no cause, however, so rapidly fatal as raw potatoes, the toxic properties peculiar to the natural order "*solanaceæ*" being seemingly exerted upon the system gene-

rally, in addition to the irritating effects of the tuber itself upon the intestinal canal.

The treatment of inflammatory or acute diarrhoea when arising from these causes becomes a matter of extreme delicacy. For, in the first place, the retention of the irritating material in the intestinal canal is a source of danger in itself; and, secondly, the extreme purgation which it induces is no less a cause of fatal termination. It therefore follows that the practitioner, on the one hand, must take care not to check the efforts of nature to expel the offending materials from the body too suddenly by the administration of powerful astringent remedies; and, on the other hand, that he should endeavour to modify the violent purgative and irritating effects by the administration of demulcents, calmatives, such as opium, and antacids, such as the bicarbonate of soda; and to support the animal's strength by diffusible stimulants, such as the ethers, wine, and alcoholic stimulants, in moderate and oft-repeated quantities; fomentations to the abdomen, and other means calculated to allay irritation and febrile disturbance, being by no means neglected, and to modify fermentation the hyposulphite of soda might be advantageously administered.

## CHAPTER LXXIV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

##### DYSENTERY.

WHILST superpurgation and acute diarrhoea most commonly attack the horse, chronic diarrhoea and dysentery are much more commonly seen in cattle, dogs, &c. Dysentery, sometimes termed "*colitis*," or "bloody flux," consists of an inflammation having a tendency to terminate in ulceration of the mucous membrane and glandular structures of the large and sometimes the small intestines. Dysentery is observed both in an acute and chronic form; the chronic in horned cattle being often dependent upon the scrofulous diathesis, with tubercular deposit and ulceration of the intestinal glands.

Dysentery in man is of two kinds—bacillary and protozoan—the former being due to either the bacilli of Shiga or Flexner, and the latter to the *Entamoeba histolytica*. It has now been found that the cat is susceptible to either of these infections, and this fact should not be lost sight of when this domestic animal is affected, on account of the extreme contagiousness of these diseases to mankind. The larger domestic animals do not appear to be affected, and are therefore not dangerous in this respect.

##### CAUSES.

Except as a concomitant of other diseases, such as rinderpest, or when induced by direct irritants, acute dysentery is a rare form of disease. It may, however, be induced both in horses and cattle by bad food or putrid water; some kinds of pastures, especially those situated upon moorlands and in



shady places ; on this account it was called "moor-ill" and "wood-evil" by the old writers. Occasionally it is seen as a complication of "red water" and "parturient peritonitis."

In the dog it is witnessed as the effect of cold and damp, and in the intestinal form of distemper.

#### SYMPTOMS.

In the ox the acute form is attended with shivering fits, variable temperature of the body, arching of the back, and increased sensibility or tenderness of the loins ; a furry tongue, and clammy mouth. The animal grunts, yawns, grinds its teeth, and at short intervals discharges a quantity of thin excrementitious material, mixed with pellets of hardened fæces and blood. There is much straining, and irritation of the rectum and anus, which appear sore and red ; some abdominal pain, evinced by arching of the back, whisking and extension of the tail, associated with tympanites, great dulness, thirst, rapid emaciation, and an aphthous condition of the mouth.

In the chronic form there is great emaciation, a verminous condition of the skin, looseness of the teeth, and anasarca swellings of the intermaxillary space ; the fæces become deeply tinged with blood, contain much mucus, and after a time an admixture of fœtid purulent matter. Ulceration about the anus ultimately appears ; the fæces are discharged involuntarily ; the eyes soon become dim and sunk into the orbit, and the animal dies.

Both in diarrhoea and in dysentery of a chronic nature the fæces contain gaseous materials, which cause the appearance of little air bubbles upon their surface when discharged from the body. The urine of an animal suffering from dysentery is of a high colour, increased specific gravity, containing an inordinate quantity of urea, proving the rapidity of the tissue changes occurring within the economy.

#### TREATMENT.

Both in the acute and chronic form much benefit is to be expected from the administration of a mild oleaginous aperient, succeeded by opium and antacids. Should these not succeed, styptics, as the oil of turpentine, or astringents, as tannic acid,

sulphate of copper, terchloride of iron, or alum; and to overcome the fœtor of the fæces, hyposulphite of soda, or other deodorizers, may be given with advantage. When the disease is essentially chronic, associated with much emaciation and a tubercular diathesis, cod liver oil in such doses as the animal will tolerate up to four ounces, given twice a day, mixed with four or five eggs, will prove advantageous. It must be remembered, however, that all treatment must be subservient to careful dieting; cake, good hay, and other articles easy of digestion and nutritive, being at all times recommendable. For the dog the same agents, in doses proportionate to the size of the animal, along with proper diet, such as rice-milk or flour porridge, are to be prescribed.

## CHAPTER LXXV.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (O.) DISEASES OF THE BOWELS—*continued.*

#### ASCITES, OR DROPSY OF THE ABDOMEN.

PERITONITIS, or acute inflammation of the coats of the abdominal cavity, having been already described in *Principles and Practice of Veterinary Surgery*, I need only refer to it here as a condition which may lead to an alteration of the peritoneal structure by the inflammation becoming chronic, and to a collection of fluid in the cavity, constituting ascites, which may be defined to be a collection of fluid in the peritoneal sac—the nature of such fluid being sometimes simple, consisting of a pale straw-coloured serum, sometimes more complex, the serosity being mixed with flakes of lymph, and inclosed in cysts or cavities by the adhesions of false membranes.

#### CAUSES.

Direct disease of the peritoneum is a very rare cause of ascites in all the domesticated animals except the ox, in which it is found frequently, as a result of tubercular growths—"grapes"—studded over the surface of the membrane.

Ascites results most frequently from diseases of the liver, the heart, or the portal blood-vessels; as a consequence of cardiac debility in some exhausting diseases; and from the ingestion of improper food, as when animals are kept during winter upon poor, ill-drained, or moorland pasture. It may also arise from anæmia and deficiency of albumen in the blood, whether induced by parasites, as in the "rot" in sheep, or independently

of such invasion from any cause inducing deficiency of albumen in the blood. It also occurs in some forms of septicæmia—braxy—and is looked upon as a condition of “crisis,” or an endeavour to excrete the morbid material from the circulation. In sheep and lambs a sanguineous form of ascites is not rarely met with, variously termed red water, water braxy, and “*maladie rouge*,” or diarrhæmia, by the French. In this latter condition, not only is there a transudation of the fluid of the blood, but also of the colouring material (see *Purpura*), the globules being sometimes dissolved, sometimes entire, and more or less altered in appearance in the serosity. Ascites is also witnessed as a complication in hydrothorax and dropsy of the pericardium.

In the dog, dropsy of the belly is generally caused by disease of the liver, induced by over-feeding and want of exercise.

#### SYMPTOMS.

These are denoted by enlargement of the belly, sheath, and sometimes the legs, the enlargement being due to effusion, as indicated by fluctuation, and a dull sound on percussion. As the fluid increases, the breathing becomes thoracic; and in the sheep and ox œdema of the submaxillary areolar tissue and inferior cervical region, with emaciation, feeble pulse, irregularity of the bowels, looseness of the hair and wool, and other symptoms common to exhausting diseases, supervene.

#### TREATMENT.

When arising from a removeable cause—as improper food—an improvement in the health and a disappearance of the collected serosity may be effected by generous diet, occasional aperients, and by tonics, more particularly the salts of iron. When associated with deficiency of albumen in the blood, it may be useful to introduce albumen into the economy by administering eggs to those in which the appetite is absent, and to those which still eat, by feeding on oil-cake and other nutritious diets, the effect of common salt as an aid to healthy digestion not being lost sight of. When ascites is due to organic diseases of the liver, heart, or other organ, treatment is of no use, except to palliate the symptoms, and in all cases it is better to put the animal out of its suffering. Paracentesis abdominis, or tapping, gives temporary, but seldom permanent relief.

## CHAPTER LXXVI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (P.) DISEASES OF THE LIVER.

DISEASES of the liver are rare in the domestic animals, with the exception of sheep, which are destroyed sometimes in great numbers by various diseases of the liver, induced by exclusive feeding upon turnips, particularly Swedish turnips, causing a fatty or degenerative condition of the organ; by stimulating food, long continued, such as the various cakes, inducing congestions and softening, and by the invasions of the "distoma" or fluke worms, giving rise to the disease termed "the rot," or of "strongyles," which appear as small bladders throughout the organ, and cause anæmia and death. But neither the horse, ox, nor dog is exempt from hepatic affections.

#### CONGESTION OF THE LIVER.

Under this head three forms are included; namely—1st. Passive congestion of the hepatic and portal veins, arising from anything which interferes with the circulation of the blood, such as disease of the heart. In my paper on *Traumatic Pericarditis*, published in the *Veterinarian* in 1857, I pointed out the co-existence of this condition of the liver. Its occurrence is explained as follows:—In heart diseases there is stagnation of blood in the vena cavæ and hepatic veins; in time, as explained by Frerichs, this is propagated to the portal vein, and to the organs from which it takes its origin; the liver then becomes turgid with blood, and the congestion extends to all the veins of the digestive system. In the same manner consolidation of

the lungs, by arresting the circulation of the blood, causes congestion of the liver and enteric veins, and is one cause of the diarrhoea which is so often an accompaniment.

The *second* form of congestion is that termed "active," in which the arterial capillaries are mostly involved. This form is induced by food of a stimulating nature, given over-abundantly, particularly during hot weather, or when the animal is not receiving sufficient exercise. It has been already pointed out that animals rapidly got up for sale frequently suffer from a diseased condition of the liver. Dr. William Budd points out how congestions of the liver occur so commonly in the human being; the same reasons are applicable to some extent to the lower animals, particularly as hepatic congestions are generally met with in over-fed, slowly worked, pampered horses, such as those belonging to brewers, or people whose business requires animals for show as well as for labour. He says—"Amid the continual excesses at table of persons in the upper and middle classes of society, an immense variety of noxious matters find their way into the portal blood that should never be present in it, and the mischief which this is calculated to produce is enhanced by indolent or sedentary habits. The consequence often is that the liver becomes habitually gorged. The same or even worse effects result in the lower classes of our larger towns from the inordinate consumption of gin and porter." Compared to its frequency in man, active congestion in the lower animals is exceedingly rare. The late Professor Coleman was of opinion that this rarity was due to the simplicity of the liver in the horse—having no gall bladder—compared to its complicated structure in man. This view is evidently erroneous, as the livers of other domesticated animals are furnished with gall bladders, and it cannot be said that they are more liable than the horse to hepatic congestions, except from errors in feeding, to which they are perhaps more frequently exposed. Hutrel d'Arboval takes another view, and describes the exemption from disease to be due to the little areolar tissue entering into the composition of the horse's liver; this view is evidently as erroneous as that of Coleman.

There is no doubt that the fact of the liver being the great filter of the body—i.e., all the food materials that pass through it on their way to the blood are deprived of their harmful

qualities by this organ—it follows, therefore, that at times an excessive amount of work has to be done, and, if the general resistance is lowered, the liver suffers by some of the organisms that gain entrance with the elaborated food finding there a resting-place. The subsequent congestion and inflammatory conditions follow in the effort to remove such organisms. It is very seldom that such a condition results in a large enough growth of organisms to promote an abscess, except occasionally in the dog.

Another cause, which is often more alarming, but never so lasting, is that form due to a sudden congestion of the liver from some outward influence, as when an animal “takes a chill.” The skin of animals, when very warm from work, is hyperæmic, and in this condition contains a large amount of blood in its capillaries. If such an animal has to stand, when so heated, in a cold, wet wind, the skin is suddenly chilled, and its capillaries contract, and as a consequence there is an immediate strain upon the internal organs in the endeavour to relieve the pressure; the liver being the largest gland and the most vascular, is naturally overloaded. An overloaded organ is unable to fulfil its functions, and the result is liver congestion, or stasis, often accompanied by alarming symptoms of high fever, followed by jaundice; and in the early stages there may be a very highly coloured urine as a result of decomposed hæmoglobin. This is especially noticeable in the tropics, where extremes of temperature are frequent. In such cases a large dose of calomel, followed by salines, is indicated.

*Symptoms.*—In hepatic congestions, as proved by *post mortem* examinations of animals which have died from “*ramollissement*,” or softening, with perhaps rupture of both liver and its capsule, traces of repeated attacks of congestion, indicated by various differences in colour and in the consistence of the hepatic textures, are not unfrequently present without there having been any manifestation of symptoms during life, beyond occasional loss of appetite, and perhaps a little dulness. Now and then, however, the following symptoms are observable:—Abdominal pain, the animal looking to the right side; yellowness of the mucous membrane; high brownish colour of the urine; constipation of the bowels;

the fæces sometimes of a clay colour and foetid, with a sour, acid, or even offensive condition of the mouth; grinding of the teeth; a desire to eat earthy substances, or lick the walls; and in very rare instances pain, manifested by lameness in the off (right) shoulder, with varying degrees of febrile disturbance.

*Treatment.*—Bearing in mind that the engorgement may become so excessive as to cause rupture of both gland and capsule, practitioners must abstract such a quantity of blood as the character and nature of the pulse will warrant; so long as the artery is round, the pulsation distinct—no matter how severe the seeming dejection and debility may be—a free abstraction of blood will be succeeded by amelioration of the symptoms, as they are but consequences of the state of hyperæmia, the removal of which being of paramount importance.

With regard to medicine, cathartics, followed by neutral salts, as the sulphate of magnesia, cause a drain from the portal system, and thus relieve the congestion. In the dog, elaterium has a special effect, by causing watery stools. A recurrence of the congestion is to be prevented by a restricted diet and regular exercise.

The *third* form of congestion is that due to engorgement with bile, arising from obstruction, parasites in the ducts, or inflammation of their mucous membrane, by which their calibre becomes diminished, and the flow of bile consequently arrested. This condition is associated with epizootic diseases, and is termed by some veterinarians “bilious influenza,” and is characterised by yellowness of the visible mucous membranes and high-coloured urine, with the symptoms of the epizootic from which the animals suffer.

The treatment of this form calls for no special comment, except that the administration of the so-called liver stimulants, as calomel, is contra-indicated, for the reason that the secretory powers of the gland are not interfered with, but that it is incapable of discharging the secreted bile, owing to the tumidity or swollen condition of the lining membrane of the small bile ducts; salines and a non-stimulating diet are therefore indicated.



## HEPATITIS, OR INFLAMMATION OF THE LIVER.

Hepatitis, or inflammation of the liver, is one of the rarest diseases affecting our domesticated animals, the majority of cases diagnosed as such being probably due to congestion. It sometimes, however, occurs, and cases are recorded where the *post mortem* examination has revealed the presence of abscesses, transformed exudates, and even ossification of the products of inflammation.

The inflammation may have its seat in Glisson's capsule, when it is called "perihepatitis," and examinations of old horses slaughtered for dissection point out the fact that this form of inflammation—the formation of false membranes, and the adhesion of the liver to the diaphragm—is of no rare occurrence, unaccompanied, however, with any symptoms during life which point to its presence. Even in the human being, perihepatitis is rarely accompanied by serious derangement, unless the inflammation extends to the portal or hepatic veins, or causes obstruction of the larger bile ducts—events of rare occurrence.—(FRERICHS.) In all probability this inflammation of Glisson's capsule accompanies the pleurisies to which the horse is so particularly liable.

Hepatitis, or inflammation of the glandular structure, may occur in a circumscribed or in a diffused form; the circumscribed leading on to suppuration—as in cases mentioned by the late Mr. John Field and others—or to the formation of patches of fibrous tissue, which appear as firm, light-coloured spots scattered throughout the organ; whilst the diffused form induces rapid degeneration of the glandular structure, with softening and atrophy of the organ, or an indurated condition of it.

In the primary stage of the acute diffused form, patches of hyperæmia occur, the peripheral portions of the lobules being filled with a more or less fluid exudate. The viscus is swollen in proportion to the number and size of the inflammatory patches; the parenchyma is loosened and lacerable. As the inflammation advances, the red colour fades, and is replaced by a brownish or greyish-red tint in some parts, and yellowish-red or pale yellow in others. This condition, as well indeed as nearly all morbid conditions of the liver, is often accompanied by a similar state of the kidneys, and sometimes of the spleen.

*Causes.*—The causes are similar to those of congestion. “In hot countries hepatitis assumes an epizootic form, especially about the end of summer. It is almost always connected with inflammation of the other abdominal organs. After death the liver is found congested, of a greyish-red colour, and weighing from forty to fifty pounds. In addition to ordinary symptoms, there is irritation of the skin. Lessona describes such an epizootic as having occurred in Italy.

*Symptoms.*—The animal is dull, inactive, has a heavy head, lustreless eye, and loathes its food. Mr. Percivall says, “the horse seems as if it was suffering some inward pain; but it is clearly not of an acute kind. It has not lain down during the past night; its dung-balls are small and dark-coloured; its urinary discharges scanty; and there is manifestly a strong fever arising in the system. The fever runs on, and commonly on the second or third day after its onset turns out to be what farriers call ‘the yellows’—known by them to be so from the remarkable circumstance of the mouth and eyes assuming that colour. The inner surfaces of the lips and cheeks, the tongue, the conjunctivæ, and in some cases the transparent cornea and iris as well, turn yellow, manifesting the diffusion of bile over the body; and the same is further demonstrated by the deep golden dye of the serum of the blood. I have also observed yellow matters floating about in the aqueous humor. The dung-balls are deeply imbued with bile, and in some cases enveloped in a viscid, bilious, mucous matter as well; their colour is that of a reddish-brown, leaving when rubbed upon white paper much the same stain as opium would. If any urine be caught, it will be found to be thick, to exhibit the same bilious tinge, and to deposit, on standing, a copious lateritious sediment. The horse will probably lie down quiet, and yet not appear easy, but from time to time turn a dolorous look at its side, and soon after raise itself up again; and if the right side be pressed against it will flinch, or bite, or otherwise express tenderness there.” In addition to the above symptoms, lameness in the off shoulder has been observed. My own experience leads me to conclude that this is a rare symptom, and is more particularly symptomatic of the existence of abscesses or of foreign bodies in the gland. In one case a thorn has been discovered; in other two, which came under my immediate observation, a stocking needle

was found in one, whilst in the other a calcified exudate, surrounded by purulent material, was detected after death. In the latter case, the symptoms of liver disease, which had continued for many months, became associated with those of chronic tetanus, and, finally, with ascites. I think it right to mention that in another animal which had died suddenly, in addition to a calcareous concretion, rupture of the liver was found, the animal having manifested no symptoms of disease during life.

*Treatment.*—In the earlier stages of acute hepatitis, purgatives and sedatives, more particularly aconite, or, if the symptoms be very acute, nauseants, as digitalis or veratrum. In human practice, ipecacuanha stands in high repute. It is given in large doses for the purpose of causing nausea, profuse diaphoresis, and frequent bilious motions. Its value in the treatment of the liver diseases of horses and cattle has been proved to be very great, and it is almost invaluable for the dog and other carnivora. It will be clearly understood that calomel and other so-called liver stimulants are inadmissible in this condition; if, however, the liver remain sluggish after the subsidence of the febrile symptoms, one or two doses may be given in combination with stomachics.

#### CHRONIC HEPATITIS.

This form of disease may occur as a sequel to or independent of acute hepatitis.

It tends to create various changes in the substance of the liver, whereby it either becomes enlarged and softened in structure, or hardened, indurated, and diminished in bulk.

Induration of the liver is described under a variety of names—cirrhosis, interstitial hepatitis, hob-nailed or gin-drinker's liver, nutmeg liver, and chronic atrophy; and the nature of the changes which occur in the liver substance are explained in three ways:—

1.) Dr. Goodeve, who takes the view of Dr. Budd, says that "when it commences with inflammation or congestion, the course which leads to atrophy is as follows:—Fibrinous exudation takes place; this occupies the portal canals, and extends even into their minute ramifications, so that the very lobules may be separated by the exudation. Livers in this early stage are much enlarged, are firm and tough—sometimes very tough—the ex-

ternal surface, perhaps, merely uneven, with commencing granulations, and the capsule more or less thickened and opaque. On section there is found considerable vascularity, an amorphous, albuminous exudation, tailed or spindle-shaped cells and fibro-cellular tissue separating the lobules. In more advanced stages the fibrous tissue is more decidedly developed. Subsequent to the formation of new fibrous tissue, contraction follows, with constriction of the vessels lying in the course of the new tissue, impediment to the circulation in the small branches of the portal veins, starvation and wasting of the tissue by them.”—(See REYNOLDS’ *System of Medicine*, vol. iii. page 345.)

(2.) According to this view it is supposed that the fibrous tissue is hypertrophied and condensed, rather by a degenerative action than by one which can be termed inflammatory. Dr. Handfield Jones says—“The change seems to be of a similar kind to that which produces cartilaginoid induration of the capsule of the spleen, stiffening of the valves of the heart, and contraction of its orifices, which can scarcely be regarded as an inflammatory origin. We are confirmed in this view by having often observed various minor degrees of condensation and thickening of the Glissonian sheaths, in cases where there was no trace of inflammatory action, as well as by a circumstance which has hitherto been quite unexplained—that is, that the spleen, albeit exposed to the backward pressure of the blood, retarded in the splenic vein, does not become distended in the way that one would expect, but is often, on the contrary, small and soft. In such spleens we have often observed very many of the nuclei throwing out fibres, which is certainly not the natural metamorphosis; and hence it seems not improbable that in this way, owing to increase of fibrous tissue in its substance, the parenchyma of the spleen is less distensible than usual, and has a contrary tendency to shrink and collapse.”

(3.) Cirrhosis is attributable to degeneration of the secreting tissue, independently of inflammation, arising from an unsuitable pabulum passing through the liver, causing a smaller demand for and diminished afflux of portal blood, wasting and absorption of the lobular structure, leaving masses of the connective tissue, which waste less rapidly than the secreting ones; this change being similar to atrophy, as it occurs in muscular tissue, and due to similar causes, namely, imperfection in the quality and diminution in the quantity of the blood.

The cases to be immediately described support this latter view, and that in all probability the atrophic change may also be partly due to the liver not being called upon to perform its function, owing to the animals having been kept in a state of semi-starvation. The cases referred to are as follows:—In the month of January 1873 Mr. James C. Dixon, veterinary surgeon, Rothbury, Northumberland, forwarded a sample of urine from a mare, requesting my opinion as to the nature of her disease. After duly examining the urine, I expressed an opinion that the animal was suffering from some affection of the liver, and wrote for a description of the symptoms. Mr. Dixon replied that he saw the mare on the 7th December 1872; it was then dull, hanging its head, and off its feed; the breathing slightly accelerated; the pulse eighty, small and feeble, but there was no cough; the breath was rather foetid; the bowels regular. In the course of a few days the pulse was down to forty-four, and it was much improved, the bowels regular; but at this time Mr. Dixon ceased visiting it. Shortly afterwards he was told that it drank too much water, and passed large quantities of urine. An examination of the food showed that both the hay and corn were very mouldy and bad. By this time it had lost much flesh, and was “awfully tucked up” in the flanks. The eyes were quite yellow, the fæces light coloured, glazed, and having a very offensive smell; the urine of a deep amber tint, and passed in great quantities. It continued alternately better and worse for some time, but losing much flesh. Mr. Dixon says, “I never saw an animal lose condition so fast in my life, except cattle when in the advanced stages of red water. After death,” Mr. Dixon says, “the liver seemed dyed with bile. I have seen grocers have coarse paper the colour of this liver.” A portion of this liver was sent to me, and found to be identical in appearance with the indurated liver of the human being. Externally the organ was more or less roughened in some places, firm to the touch, and broke down under the finger less readily than natural; in fact was firm, dry, tough, and fibrous, and on section the lobules were in parts completely replaced by a white fibrous tissue, and in others surrounded by an increased quantity of the same structure, giving to the cut surface a mottled granular appearance similar to that presented in the interior of a nutmeg.

The kidneys of the same animal were in a stage of degeneration.

Two other animals died on the same farm, namely, a mare and a six months' foal, between the time mentioned and the 25th of March, from liver and kidney disease. The symptoms and appearance of the mare are described by Mr. Dixon as follows:—"The owner says that for ten days or so before it left off feeding it was very dull, and took a deal of driving. It left off feeding on a Thursday, and I was sent for on the following Saturday, when it presented the following appearance and symptoms:—Standing with head depressed; dull and oppressed; eye heavy, and eyelids partially closed; resting from one hind foot to the other now and again; ears, legs, and body of a

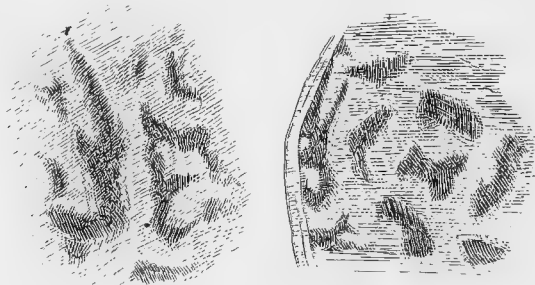


FIG. 82.—Cirrhosis of the Liver.

The white parts consist of white fibrous tissue surrounding the atrophied gland structures, slightly magnified.

natural heat; the pulse small and weak; the breathing slightly increased (the pulse and breathing very like what we find in influenza); fœtid breath; bowels easy and natural; the urine rather high coloured. In a few days its pulse came down to sixty, and continued to do so gradually for ten days until it came down to forty-four, at the same time increasing in strength. I did not see it again for ten days or so, and in the course of eight days it got decidedly worse, and was now very thirsty and passing urine very often; pulse agitated, and fifty-six; fearfully tucked up in the abdomen; lost condition terribly; anxious for water; bowels now confined; fæces light in colour and covered with mucus; eye quite yellow; membrane in the space between the last incisor and first molar of the upper jaw yellowish in tinge; breathing increased; evinced pain on pres-

sure on the false ribs on right side up and down, anteriorly and posteriorly. Next day pulse down to forty-four; anxiety for water and frequent staling gone; and continued thus for about ten days; eating well; was more lively; the breathing more gentle; the bowels kept regular. By the end of this time it fell off again, and shortly after it improved, and was better upon the whole; but the urine was now passed in a small stream, and instead of being amber-coloured was of a reddish-brown appearance; evinced no uneasiness when voiding it; continued to eat in a fair way for a week or so. By this time its eye was not so yellow; it left off feeding, and its pulse was now of a very different character—sixty, rather full artery, and rather wiry; it gradually sank, and died quickly. It never coughed during the time it was ill, and it lay on both sides. The eye at first was not yellow, but to me appeared blanched.”

Mr. Dixon also sent me the symptoms presented by the foal. He says—“I was called to the foal on the 13th of March (but I saw this foal in December, when it was a nice, thriving, promising animal), but about eight days before I was called to it I was passing the farm and looked in. The foal was in a box. I went in and handled it, and I was perfectly struck by its appearance—just shaggy hair, skin and bone; the hair of a dirty dry colour, in fact quite darkened to a mousy brown, while before it was a lightish brown; pot-bellied. I examined the hay in the rack, took out a handful of dry, musty, black stuff, more fit to litter pigs; so I was called on the day mentioned. It seemed inclined to hang down its head, or oftener stood with it in the manger, resting on the under jaw; fore legs apart; pulse eighty, with some degree of power in it; eye yellow; the pot-belly gone, and rather tucked up; pain on right side (like the rest). Next day comatose, pushing its head in corner of box, cold legs and ears, and dead next morning. Great debility and weakness were present from the first. I may add that the foal had never refused eating until the day I was called to it; it ate none after that, and it was not confined in the bowels.”

It appears from the history of these cases that cirrhosis of the liver was due to a gradual starvation, and that the atrophy of the hepatic secreting structures was part of the general emaciation—atrophy—of the whole body, which was so rapidly going on, consequent on want of proper food.

Cirrhosis, consisting as it does of an organic change in the gland, tends to a fatal termination, but the rapidity of its course and progress may in some instances be modified by the removal of its cause.

It may be observed that the same condition of the liver has been discovered to prevail as an epizootic in Nova Scotia, under the term of the Pictou disease, and on the Continent of Europe as lupinosis; and Mr. D. Hutcheon, the Chief Veterinary Surgeon to the Cape Colony, says that similar conditions of the liver are common in the colony, as a consequence of periodical times of semi-starvation—when the grasses are scarce—succeeded by sudden repletion during the rapid growth of the said grasses in the rainy seasons.

#### SOFTENING—RAMOLLISSEMENT—RUPTURE OF THE LIVER.

Softening and enlargement of the liver is due probably more to repeated attacks of congestion or engorgement than to a truly inflammatory change.

Softening of the liver, very often without there having been any indications during life, terminates in sudden death from rupture of the gland and its capsule. In some instances, however, there are certain symptoms of disease of the organ, such as pallidity or yellowness of the visible mucous membranes, occasional attacks of what appears to be colic, nausea, and irregularity of the bowels.

These attacks of apparent illness usually subside in a short time, leaving the animal seemingly in perfect health. They are, however, apt to recur, the patient finally rapidly sinking from what at first appeared a slight attack of colic, or from some sudden or violent exertion. Death does not always immediately occur from rupture, as it would appear that the liver may become repeatedly ruptured without the occurrence of a fatal hæmorrhage. So long as Glisson's capsule remains intact, the hæmorrhages are not fatal; and even rupture of the capsule itself, when limited in extent, is not always followed by immediate death. In a case mentioned by Mr. Siddal (Percivall, vol. ii.), a horse had been ill, and subject to frequent fainting fits for upwards of three weeks before it died, which appeared afterwards to have been caused by small ruptures of



the peritoneal coverings of the liver in different places, from all which it rallied, not sinking until the grand rupture had happened. I have met with cases which, remaining dull and off their feed after an attack of colic, have suddenly sunk; and *post mortem* examination has revealed the existence of several small ruptures, and the final one, of larger size, from which several gallons of blood have escaped into the peritoneal cavity. When small ruptures occur, the hæmorrhage becomes arrested by coagulation of the escaping blood; this may become encysted by the formation of a fibrous membrane round it, the extravasated blood becoming shrunk by absorption of its fluid parts, collections of fine crystals of hæmatin being left.

The symptoms of approaching death are, fainting fits; violent colicky pains; the animal breathing heavily, looking round to its right side; there is rapidly increasing pallor of the mucous membrane; coldness of the mouth, and of the body generally; dilatation of the pupils; the animal propping itself on its limbs, or by the side of the stall; and a running down pulse. If these symptoms be preceded by yellowness of the mucous membrane, by tinged urine, or clay-coloured fæces, they are diagnostic that the hæmorrhage proceeds from rupture of the liver.

*Causes.*—The causes which induce softening and disorganization of the liver are identical with those inducing congestion, namely, stimulating and fattening foods, particularly linseed-tea and linseed-cake, or want of sufficient exercise; and animals which die from this lesion are generally fat and sleek in the coat.

It is stated that the liver may become ruptured by kicks or blows and severe exertion. This seems to me, however, very improbable, without there being a previous disorganization of the organ.

*Post mortem appearances.*—On cutting into the abdomen a large quantity of a semi-coagulated dark-coloured blood will be found in the peritoneal sac; if due to oily food, globules of oil will be seen floating in it; the tissues of the body are blanched, and generally intermixed with much fat. With regard to the liver itself, it is found to present two pathological conditions: *first*, highly congested, enlarged in every direction, its vessels filled with dark semi-fluid blood, and its tissues with much serosity; in other cases only shreds

of the capsule, and small portions of liver substance may be found.

Now and then various parts of it present a peculiar mottled appearance, the centre of the lobules being of a dark red colour, whilst their peripheral portion is of a yellowish-white; this condition constituting what is generally termed the "*true nutmeg liver*." Sometimes the organ has acquired the enormous weight of sixty pounds or more.

In the *second* form, we find that the liver is not so greatly enlarged as in the first, its capsule presents a peculiar slate-coloured tint, and the whole organ may be of the same hue, or stained deeply yellow. Both the gland substance and the capsule are friable, easily reduced to a pulp if touched with the finger, and having more the consistence and appearance of moist, bluish, or yellowish clay than of an organized structure.

Whilst horned cattle, and particularly sheep, are liable to suffer from disorganization of the liver, they seem to escape fatal termination by rupture. Sheep, however, die in great numbers from liver disease about the period of parturition; sometimes without presenting any symptoms of liver disease, or of any disease whatever; sometimes anæmic, and occasionally with well-marked symptoms of jaundice.

It is impossible to account for these varieties in the symptoms when the pathological conditions of the organ are identical; and we can only accept them as facts which further researches may throw light upon.

*Treatment.*—This can be only prophylactic. An inquiry into the causes will show that the errors which lead to the gradual disorganization of this organ are those of improper feeding. In the horse and ox, food highly charged with nutritious elements, and mixed, or but slightly mixed, with that of a coarser nature. In sheep, as already pointed out, food containing saccharine, and but little nitrogenous materials, as when they are fed exclusively upon turnips.

It is therefore necessary, in order to prevent these various alterations of structure, that the proper dieting of animals should always be attended to; that when sheep are put upon turnips, nitrogenous food should be allowed them throughout the whole turnip season in some shape or other, as hay, straw, or small quantities of cake, or bean meal.

## JAUNDICE—ICTERUS, OR THE YELLOWS.

Although spoken of as a disease, jaundice is in reality but a symptom of many affections in which the tissues of the body are dyed yellow. Although a result of many organic diseases of the liver, it sometimes occurs when that organ is healthy, and is not rarely absent when it is in a state of disorganization. It may be produced artificially by ligature of the common duct, which proves that obstruction of the flow of bile into the intestine can cause the disease. It may also occur when there is no such obstruction. And to account for these varieties of causes various theories have been advanced:—*First*, that the bile is formed in the blood, and is merely removed by the liver, and that jaundice is a consequence of non-separation of the bile; *second*, that in some diseases the hæmatin of the blood is changed into bile-pigment, with disintegration of a large number of blood corpuscles, thus assigning a blood origin to the colouring matter of jaundice; *third*, that it arises from abnormal diffusion of bile, proceeding from some alteration in the circulation of the blood in the liver, or else to defective metamorphosis or impaired consumption of bile in the blood. It is now, however, generally admitted that some of the ingredients of the bile are generated in the liver—namely, the bile acids, glycocholic and taurocholic acids found in the bile combined with soda bases; whilst others—the bile pigment, biliverdine, bilirubin, or cholepyrrhin and cholesterine—exist in the blood, and are merely separated by the liver in a manner similar to the separation of the urea from the blood by the kidneys. It is now admitted that jaundice may arise from two distinct causes:—*1st*. Suppression or non-elimination; and *2d*. From reabsorption of bile.

The two forms are distinguished by the presence of the biliary acids in the urine when it arises from reabsorption, and their absence when due to suppressed secretion. The test is as follows:—To a couple of drachms of the suspected urine add a small fragment of loaf sugar, and afterwards pour slowly into the test tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present there will be observed, at the line of contact of the acids and urine, after standing for a few minutes, a deep purple hue. This result may be taken as a sure indication

that the jaundice is due to obstructed bile ducts. On the other hand, the absence of this phenomenon, and the occurrence of merely a brown instead of a purple tint, although in the earlier stages of jaundice equally indicative of suppression, is no indication of the cause of the suppression, which must be gleaned from other circumstances.—(HARLEY on *Jaundice*.) Non-secretion of bile may arise from a variety of causes:—1st. Innervation; 2d. Disordered hepatic circulation; 3d. Absence of secreting structure, as in atrophy, the invasion of tubercle, and the degenerations.

Jaundice from reabsorption is arranged as follows:—1st. Obstruction by foreign bodies within the bile duct; 2d. Obstruction by inflammatory tumefaction of the duodenum or of the lining membrane of the duct, with exudation into its interior; 3d. Obstruction by stricture or obliteration of the duct; 4th. Obstruction by tumours closing the orifice of the duct or growing in its interior; 5th. Obstruction by pressure on the duct from without; 6th. Obstruction by parasites.

Looking upon jaundice, which is manifested by yellowness of the mucous membranes, as symptomatic of disease, it will be necessary, before the practitioner prescribes any particular treatment, that he should examine the urine in order that he may determine whether it arises from any cause obstructing the flow of bile into the intestinal canal, or from a disordered condition of the liver itself, consequent upon which true bile is not secreted.

*Treatment.*—In jaundice from suppression, if the cause of the suppression be congestion or inflammation of the liver, the treatment recommended for those conditions is to be pursued. If, on the contrary, those conditions be absent, liver stimulants, as calomel with aloes, may be administered. In the dog, podophyllin, with hyoscyamus; or benzoic acid, from 10 to 15 grains, divided into three doses, daily.

As repeated cathartics are not admissible in the lower animals, benefit is often derived from taraxacum, nitro-muriatic acid, and in some cases ox gall. It must, however, be understood that no permanent benefit may be expected if the jaundice be due to cirrhosis or the degenerations. The animal will, however, continue to perform moderate work, provided it be properly and carefully dieted.

In jaundice from reabsorption, the cause of obstruction must be inquired into. If it be due to catarrhal inflammation of the mucous membrane, purgatives, more especially in the horse, are contra-indicated, the constipation of the bowels being more safely removed by mild aperients, such as four-ounce doses of the sulphate of magnesia, or half a pint of linseed oil administered daily until the fæces become pultaceous. The debilitated state of the liver—which sometimes remains after acute attack, and in which the organ performs its functions irregularly, sometimes secreting inordinately, at other times scarcely secreting at all; the inordinate secretion being characterised by bilious purging, some portions of the fæces being coffee-coloured, tinged with altered bile; and the non-secreting condition by clay-coloured fæces with fœtor—is to be overcome by tonics, more especially iron, cinchona bark, or quinine, and a carefully regulated diet. Should the animal indicate, by licking the walls or grinding its teeth, that the stomach and bowels are in an acid condition, great relief will be afforded by the antacids, lime water, or the carbonates of soda or magnesia.

It may be observed that the cause of obstruction will, if long continued, induce such organic changes in the liver itself as to destroy its secretory function; hence we find in such diseases as the "rot" in sheep that the biliary acids are not present in the urine towards the later stages.

In dogs suffering from jaundice and ascites, elaterium, by inducing watery stools, often gives temporary relief, and prolongs the life of the animal.

NECROTIC ABSCESES are rather frequently found in the liver particularly in the livers of young cattle. They rarely give any indication of their presence during life, the animals dying apparently from some mysterious cause. They are either isolated or, rarely, in groups, and are often supposed to be tubercular, but upon microscopic examination the tubercle bacilli are found to be absent, and there is no appearance of successive growths, as in tuberculosis. Each abscess is surrounded by a capsule or zone of condensed hepatic tissue of a dark brown colour, surrounding a mass of broken-down fibrinous material having a yellowish or sometimes brownish-white appearance.

These abscesses are caused by the bacillus of necrosis. A long, thread-like, filamentous organism, very minute. Gram negative, and difficult to stain in section. It may be demonstrated microscopically from the edge of the lesion, where it grows freely, causing an ever-increasing necrotic mass. This is the same organism that is associated with the necrotic lesions occurring in the lips and nostrils of calves, and at one time thought to be diphtheria. The organisms are probably introduced from the digestive tract, and absorbed into the liver, which they most frequently attack, the bacillus of necrosis being often an inhabitant of the mouth in adult cattle and causing apparently no harm. Spontaneous recovery may take place, and the healed lesions may sometimes be seen as old scar tissue in animals slaughtered at abattoirs.

In my opinion many of these supposed abscesses are broken down thrombi or emboli, consequent upon a mild attack of omphalitis during early life, remaining dormant for an indefinite period, but from some cause or other becoming liquefied, and thus infect the blood stream, inducing death by septicæmia.

GALL STONES are very rarely found in the lower animals, and there are no symptoms indicative of their presence during life, beyond those which may be induced by any disease of the liver itself. A case in the horse was recorded by Mr. Faulkner, of Manchester, in 1896, and many gall stones, varying in size from a pea to a hazel nut, were found on *post mortem* examination. Another case is recorded by Mr. Thompson, of Aspatia, in which he found a large gall stone when making a *post mortem* on a horse. Only recently Mr. H. Sumner, of Liverpool, found a liver at the knackers', in the bile ducts of which there were innumerable small gall stones. None of these cases were diagnosed during life. It may, however, be mentioned that in horned cattle which have died from any exhausting disease, with prolonged absence of appetite, the gall bladder is generally distended with an inspissated bile, in which much sediment may be found adhering to the mucous membrane, and that in sheep deposits are found when the gall ducts have been dilated by "flukes."

## CHAPTER LXXVII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (Q.) DISEASES OF THE SPLEEN AND PANCREAS.

*Diseases of the Spleen.*—Various organic changes, as atrophy, hypertrophy, thrombosis, tubercle, cancer, the presence of hydatids, lymphadenoma, and ossification are found *post mortem*; but there are no symptoms during life which indicate their presence. The late Mr. Walter Lewis, of Crewe, mentions that in two cases of diseased spleen there was a tendency to run backwards in the stall and elsewhere. I have already stated that I have witnessed this symptom in disease of the duodenum (see *ante*, page 804.) I am informed that in America the horse suffers from an intermittent fever in which the spleen becomes enlarged. It is stated that enlargement of the spleen may be detected *per rectum*. I doubt, however, its possibility, unless, indeed, the spleen be of an enormous size. In one case which came under my own notice, lymphadenoma of the spleen (shown in the frontispiece of the first edition of this work) was guessed at by the absence of signs of disease of any other organ, slight increase of the white globules in the blood, pallidity of the mucous membrane, a stiffness of the back, and a gradual wasting of flesh. In another case lymphadenoma of the spleen was diagnosed from the history of a case which had a lymphadenomatous tumour in the parotidian region. This animal had latterly fallen off its appetite, and was subject to slight recurring colicking pains, but there were no other symptoms of internal disease. I removed the tumour from the neck, but after finding out its histological character, gave an unfavourable prognosis, being of opinion that another of the same nature was in existence,

most probably in the spleen, giving rise to the colicky symptoms. The animal died a few days afterwards, and a hard lymphomatous tumour twenty-five pounds weight was found in the spleen, involving walls of stomach, duodenum, &c.

I carefully examined the blood of this animal, and found there was scarcely, if any, increase in the white corpuscles (leukæmia), a condition sometimes associated with hyper-development of lymphatic tissue.

I have met with several cases in which the lymphatic glands in various parts of the body, but more particularly in the cervical and subcapular regions, have been enlarged, with development of lymphatic tumours in the spleen and liver.

The same kind of growth is also found associated with farcy, and in the case from which the illustration was obtained the liver weighed thirty-five pounds, being studded throughout with the white tumours shown in the figure.

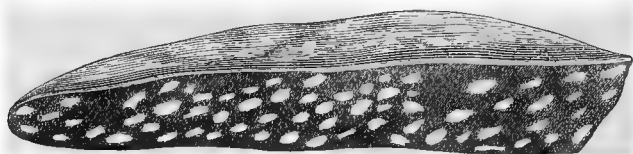


FIG. 83.

The development of lymphadenomatous growths, although found associated with farcy, is not to be considered as due to the presence of the glanderous poison, as they frequently occur when there is not the slightest suspicion of malignant disease.

Lymphadenoma, for a long time confounded with tubercle, differs very materially from that product; in fact, whereas tubercle rapidly undergoes degeneration, lymphoma has little or no tendency to retrograde. Microscopically lymphoma consists of a delicate network of fibres, within the meshes of which are contained numerous cells—lymph corpuscles. In the early stage of the growth the number of cells is very great, and many of the cells may contain two or even more nuclei, but in a more advanced stage the proportion of cells is smaller, and the reticulum forms most part of the growth.

The physical characters, according to Green, vary according to the rapidity of the growth. The rapidly growing forms, in



which the cellular elements are numerous, are of a greyish colour and soft brain-like consistence, much resembling encephaloid cancer. These often attain an enormous size, and infiltrate the neighbouring structures. They have been called by Virchow lympho-sarcoma. Those which are more slowly developed, and in which the reticulum constitutes the greater portion of the growth, are much harder in consistence, sometimes being almost cartilaginous.

I have never witnessed the lympho-sarcoma of Virchow. In the case from which the figure was drawn, the tumours, although in the aggregate weighing so heavy, were individually no larger than a thrush's egg, and were moderately firm in consistence.

The origin of these tumours is a matter of obscurity. I have seen them in the well and in the ill fed animal.

The above remarks will apply to diseases of the pancreas. It must be, however, held in remembrance that the pancreatic juice has the property of converting the fatty matters of the food into an emulsion. The presence, then, of fat or oil in the fæces of the dog, or of any other animal of which fat is an ingredient in its food, points to some diseased condition of the pancreas. A remarkable fact is often observed on making *post mortem* examination of horses which have either been slaughtered on account of age or incurable lameness, or have died from some ordinary disease not associated with pancreatic disease, and that is that the major portion of the pancreas has disappeared, and only shreds of it can be detected.

Lymphadenoma shows symptoms almost identical with leukæmia, and the treatment is the same, but results are not good. The swelling of the sub-maxillary gland may be confounded with glanders, but the regular swelling on both sides and other chains of similar gland swellings will dispel this idea. There is no leucocytosis, and the general debility is rapidly progressive.

## CHAPTER LXXVIII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (R.) DISEASES OF THE KIDNEYS.

OWING to the fact that the lower animals are free from mental emotions, the cares and troubles of the world, and that they do not indulge in alcoholic drinks, the kidneys are in a great measure exempt from those diseases which so often destroy human life.

Diseases, however, do occur with which the veterinarian has to cope. Physiologically, the kidneys excrete from the body those materials resulting from metamorphosis of tissue which would, if retained, act injuriously upon the system generally; and the secreted material—the urine—differing as it does in various animals, contains certain constituents, some of them identical, some dissimilar. The presence of these constituents in normal or abnormal quantities, or their absence, indicate to the investigator various morbid conditions of the kidneys, as well as many changes which occur in the body during disease. The urine is naturally alkaline in the herbivora, and acid in flesh-eating animals. It consists of a large amount of water; a nitrogenous substance called urea; an acid—hippuric in that of herbivora, and uric in the carnivora; colouring matter called uro-hæmatin; inorganic salts; organic substances of an ill-defined nature called extractive matters.

The density of healthy urine of the horse appears to range from 1030 to 1050; that of the ox has a specific gravity ranging from 1032 to 1040; that of pigs 1010 to 1012; and of the goat 1008 or 1009.—(VON BIBRA.) The amount of water in the urine varies much, according to the quantities of fluids that have

been taken into the system, and the condition of the intestinal canal, skin, and the surrounding atmosphere.

The amount of urine daily excreted by healthy horses varies, according to the observations of Colin, from 44·033 to 61·646 English pints; and the amount of water in 1000 parts of urine, according to the analyses of Von Bibra, Boussingault, and others, from 880 to 930 parts; whilst in that of the ox, Von Bibra found 912·01 and 923·11; in that of the pig from 980 to 990.

Urea is the most important product of tissue change; it has the same chemical composition as the carbonate of ammonia, to which it is readily converted when heated a little above the temperature of boiling water, or when kept in contact with decaying animal matter. When pure, it crystallizes from a watery solution as a white, semi-transparent, crystalline body with a bitter taste. The crystals are quadratic prisms, with rectangular terminal planes; it readily combines with nitric acid, forming the nitrate of urea, which speedily crystallizes; and the addition of nitric acid to urine is the method of detecting its presence in abnormal quantities. The crystals of the nitrate of urea, when rapidly formed, are flat, shining, rhomboidal plates (see fig. 56, p. 560); when slowly crystallized, fine prisms. Nitrate of urea is soluble in water and alcohol, but only sparingly so when they contain an excess of nitric acid; it is insoluble in ether.

It combines also with oxalic acid, forming the oxalate of urea ( $\text{Ur. C}_2 \text{O}_3 \text{H O}$ ), which crystallizes in prisms and quadrilateral tubes. Urea forms insoluble compounds with the nitrate or protoxide of mercury.

Although found in the urine, urea is not formed in the kidneys, but is excreted by them from the blood; and, as already pointed out in former chapters, it results not only from metamorphosis of tissue, but also from excess of food, as well as from the decomposition of uric and hippuric acid. Its formation from uric acid has been experimentally proved by Neubauer, who on giving rabbits from 31 to  $46\frac{1}{2}$  grains of uric acid with their food, the quantity of urea excreted in the twenty-four hours was augmented from 32·5 to 65·1 grains. Uric acid, when acted upon by permanganate of potash, is transformed into several substances, one of which is urea.

The elimination of urea is also influenced by water. The more water an animal drinks the more urea will it excrete. Common salt also produces an augmentation in the excretion of urea.—(BÖCKER, BISCHOFF.) Certain foods diminish the daily excretion of urea, such as sugar, starch, fat; in fact, says Dr. George Harley, "my idea is that all nitrogenized foods augment, and that all non-nitrogenized foods diminish, the amount of urea eliminated by the kidneys."

Increased elimination of urea occurs in all febrile diseases, and is indicative of abnormal metamorphosis of tissue; and its amount bears a close relationship to the exaltation of temperature and intensity of fever. Retention of urea is a very unfavourable circumstance, and is symptomatic of intense congestion of the kidneys, exudation and hæmorrhage into the uriniferous tubes, and desquamation of their epithelium, with destruction of their secreting cells.

Uric acid,  $C_{10} H_4 N_4 O_6$ , is a white crystalline, feebly acid, tasteless, organic substance, insoluble in ether or alcohol, and but sparingly so in water: 2000 parts of hot and 11,000 parts of cold water are required to dissolve one part of uric acid. It is soluble in strong sulphuric acid, and is transformed by dry distillation into urea, cyanic acid, hydrocyanic acid, carbonate of ammonia, and an oily coal. It unites with bases such as potash, soda, or ammonia, forming with them crystalline urates. The urine of herbivorous animals contains little, if any, uric acid; but it is found in the solid urines of serpents, birds, and insects, and in the liquid urines of omnivora and carnivora, in combination with potash, soda, and ammonia.

The pathology of uric acid is of little consequence to the veterinarian. It is derived from the same source as urea, and whatever accelerates oxidation increases the amount of urea and diminishes the uric acid, and whatever diminishes oxidation decreases the urea and increases the uric acid in the urine, thus proving that urea is a more completely oxidized product than the uric acid. In the dog and other carnivora uric acid calculi have sometimes been found.

Hippuric acid ( $C_{18} H_9 N O_6$ ) is a beautiful white crystalline, slightly acid body, constantly found in the urine of all but the purely carnivorous animal. It is soluble in 400 parts of cold water, and its solution is sufficiently acid to redden litmus

paper; it is also soluble in ether and alcohol. Its quantity in the urine varies greatly. When the quantity of urea is small that of hippuric acid is almost always large, and *vice versa*. According to Von Bibra and Boussingault it varies from 5 to 15 parts in 1000 of the urine of healthy horses; and it has been stated by some chemists that the urine of horses which are subjected to very hard work contains no hippuric acid, but much benzoic acid instead. "The last part of the statement," says Mr. Gamgee, "seems doubtful, though it would appear that hard work checks the excretion of hippuric acid, and increases the quantity of urea excreted." The observations of Maack oppose this conclusion. He found that horses kept standing in the stable passed very little hippuric acid, but large quantities of urea, and Dr. G. Harley made similar observations regarding London stall-fed cows. They did not, however, examine the urine after these animals had been exercised; their experiments are therefore incomplete. Hippuric acid is increased in rheumatism, red water, and in diseases of the respiratory organs; in fact, in all conditions in which the blood is imperfectly aerated, either from obstruction to free respiration or sudden arrestment of the cutaneous functions, as well as from an imperfect condition of the blood itself, rendering it incapable of oxidation.

Extractive matters, as creatine, creatinine, lactic acid, and benzoic acid, which is transformed into hippuric acid in the body—for it has been found that when benzoic acid is given to an animal an almost equal amount of hippuric acid is eliminated by the kidneys—are found in the urine. Sometimes urine contains a large quantity of mucus derived from the pelvis of the kidneys, ureters, bladder, or urethra, indicating some degree of irritation of the mucous membranes. After parturition, as might be expected, the urine generally contains much mucus, derived from the mucous membrane of the vagina.

Saline matters, consisting of various materials from the food and tissue, as the chlorides, phosphates, sulphates, and carbonates, are present in healthy urine, whilst other salts, originating in mal-nutrition of tissues, or from defective secretion of other organs, such as the oxalates, are sometimes met with. For the method of determining the presence of these, the reader is referred to Harley on the *Urine*, Roberts on *Urinary and Renal Diseases*, Beale, and others. The most common inorganic deposit which

is found in the urine of the horse is the carbonate of lime, which appears as a chalky sediment if the urine be kept in a vessel for some hours.

In the ox and sheep, when fed upon turnips and linseed cake, the urine generally becomes highly charged with the phosphates, which are very often spontaneously precipitated upon the mucous membrane of the bladder, urethra, and the long hairs surrounding the prepuce. I have some specimens in my possession of the ammonio-magnesian phosphate in the form of tubes deposited upon the hairs surrounding the preputial opening. The occurrence of phosphatic deposits in the lower animals results from the food upon which they are kept being highly charged with phosphatic salts. We consequently find that when animals are fed upon turnips and other foods grown upon land highly manured with dissolved bones and other phosphatic preparations, that the urine becomes highly charged with these materials, and that they are apt to be deposited in the form of gravel upon the mucous membrane of the urinary passages, more especially upon the occurrence of any cause of irritation, such as a common cold or other catarrhal affections. They sometimes induce obstruction to the flow of urine if situated in the urethra, or a suppression of its secretion, either by extension of the irritation primarily excited in the urethra or bladder to the kidneys, or when deposited in the pelves of the kidneys by an inflammation of the glands. In order to overcome the further deposition of this salt a change of diet is essential, and to dissolve what is actually deposited, nitric acid in fifteen-drop doses, largely diluted, may be administered three times a day.

Pus or blood are occasionally met with in the urine, and indicate irritation or inflammation of the urethra, bladder, or kidneys. When from the urethra, bladder, or ureters, the extravasated blood has no definite form, but if from the kidneys, blood and lymph are moulded into the shape of the uriniferous tubes.

*Albumen* is recognised by its property of coagulating upon the application of heat and of nitric acid. If the urine be alkaline the albumen may not coagulate upon boiling, but if a few drops of nitric acid be added it is immediately precipitated. Sometimes, if the urine be boiled, a precipitate may be formed, consisting of the phosphates; these are dissolved by nitric acid.

Again, if the urine contain much urea, a precipitate is formed when nitric acid is added ; this is, however, dissolved if the liquid be boiled, and the determining test for albumen is that it is neither dissolved by heat nor nitric acid. The presence of albumen in the urine is diagnostic of congestion, inflammation or degeneration of the kidneys, or that condition of the system already described under "Red Water." Albumen is also present in the urine in some cases of indigestion, and indicates that the albumen of the serum is in a condition which renders it unfit for the nutrition of tissues.

The occurrence of oxalic acid in the urine has already been referred to (see *Oxaluria*).

#### DIABETES MELLITUS,

or saccharine urine, or *Glycosuria*, is seldom met with in other of our patients than the dog.

When an animal is so affected, the symptoms noted are dulness, weakness, and gradual emaciation ; if the disease be in its initial stage, there is intense thirst, a voracious appetite, and excessive secretion of urine.

The specific gravity of the urine is increased to 1060 or more, and sugar can be recognised by employing Fehling's test.

As the disease progresses, soft cataracts appear in the eyes, and the animal in the majority of cases dies of coma.

There are no special *post mortem* lesions discoverable except, perhaps, fatty liver and general emaciation.

The following are the normal specific gravities and daily quantities of the urine of domesticated animals in health and under usual conditions as regards feeding and watering :

#### NORMAL URINE.

			Specific Gravity.	Quantity per Day.
Horse	...	...	1036	8 to 10 pints.
Ox	...	...	1007 to 1030	2 to 2½ gallons.
Sheep	...	...	1006 to 1015	1 to 2 pints.
Pig	...	...	1003 to 1025	3 to 12 pints.
Dog	...	...	1020 to 1040	½ to 2 pints.

It is necessary, if one desires to make a correct examination of urine, to measure the whole amount excreted in twenty-four hours.

In veterinary practice the following are the more important substances found in the urine, and indicative of some disease: *Albumen, urea in excess, bile, blood, pus, organisms, oxalates, phosphates, glucose, cystin, mucus, casts from the kidney (hyaline, epithelial, and blood)*, and appended are the methods of detecting the commoner ones.

*To detect albumen in the urine.*—1. Boil some urine in a test-tube. If there be a precipitate, it indicates albumen or salts. Citric acid and nitric acid dissolve the salts, but not the albumen. 2. To urine in a test-tube add drop by drop a concentrated solution of *picric acid*. If a cloudiness appear, and does *not* disappear on heating the mixture, then albumen is present.

*Blood* may be detected microscopically by means of the spectroscope or by Heller's test. To 10 c.c. of urine in a test-tube add caustic soda till the mixture is strongly alkaline. If blood be present, when the mixture is boiled the deposit of earthy phosphates is brownish-red, owing to the presence of hæmatin, and the fluid in the tube is bottle-green in colour.

*Sugar (Fehling's test).*—Render the urine alkaline with caustic potash, and filter. Fehling's copper solution (which should be freshly prepared) is then added to the mixture. If glucose be present, cuprous oxide is precipitated. In suspension, this varies in colour from a light green to yellow, or to a reddish-brown, according to the quantity of sugar present.

*Bile.*—Pour some weak solution of methyl violet down the side of a test-tube containing the suspected urine. If bile be present, a bright carmine ring will appear at the junction of the two fluids.



## CHAPTER LXXIX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (R.) DISEASES OF THE KIDNEYS—*continued.*

#### INFLAMMATION OF THE KIDNEYS—NEPHRITIS.

A RARE disease in the lower animals, but in human medicine it is described under three heads, namely—*Suppurative, Interstitial, and Tubal.* Except from the irritation of a calculus, suppurative nephritis—pyelitis—is a very rare form of disease in the lower animals. I have, however, met with a few cases where it has arisen independently of such source of irritation. In one case (a cow) it was associated with inflammation of the urino-genital mucous membranes, consequent upon difficult parturition; indeed it seemed to be due more to the absorption of infective materials from the inflamed mucous membranes, which discharged a fœtid, purulent material very profusely, than to an extension of the inflammation. The next case was that of a foal two days old, which had died from acute suppurative arthritis. A *post mortem* examination revealed not only the presence of pus in the tissues surrounding the articulations, but sundry purulent points in the kidneys, the pus in which being more or less inspissated or caseous.

I look upon this case as important, as to some extent it supports the view that “joint disease” occurring in young animals is not always a primary disease, but secondary to the formation of pus in other parts of the organism. Generally, however, nephritis embraces all the structures of the kidneys, often commencing in the mucous membrane of the uriniferous tubes, afterwards involving the parenchyma, and terminating in resolu-

tion, or in desquamation of the lining of the epithelium and degeneration of the secreting structures, sometimes in suppuration, and in very rare instances *gangrene*.

*Causes.*—Nephritis is said to occur from injuries, such as blows and strains. My experience leads me to think that this idea is exaggerated, and that it occurs, and that but rarely, from the internal administration of irritant diuretics, as turpentine, the resins, or cantharides, the absorption of cantharidine from large blisters, the irritating effects of croton oil when given as a purgative, and still more rarely from the effects of stimulating food, or of the long-continued effect of cold, such as cold water dropping upon the animal's back, directly applied to the loins. Many ailments and injuries are mistaken, I think, for nephritis, more especially if they are characterised by a straddling or stiff gait.

*Symptoms.*—There is considerable fever and colicky pains; indeed the malady closely simulates colic with fever. There is a hard, frequent pulse; increased thirst; short, rapid breathing; hot, clammy mouth, and constipation of the bowels. There may be some stiffness of the loins and disinclination to move, but it by no means follows that stiffness, a straddling gait, tenderness of the loins, arched back, are constant symptoms; indeed, I am led to conclude that they are present only in the minority of cases, and that the only signs by which the true nature of the disease can be determined are a scanty secretion or total suppression of urine, desire to micturate frequently, the animal stretching itself in vain attempts, passing perhaps but a few drops of a concentrated, highly coloured, and irritating secretion, which, if carefully examined microscopically, will be found mixed with sanguineous or fibrinous casts of the uriniferous tubes, blood globules, epithelium, and even pus cells. If tested by heat or nitric acid, a more or less abundant precipitate of albumen will be thrown down. In those instances where there is total suppression of urine, the application of the determining test is impossible. The true nature of the disease may, however, be arrived at, from the fact that when fever, with colicky pains, is present, in addition to emptiness of the urinary bladder, frequent attempts to urinate, but no urine being discharged, that the non-secretion is due to inflammation of the kidneys, which are unable to perform their functions in consequence of the inflam-

matory process. Cases now and then occur in which the only symptoms present are suppression of urine, with fever; but in other instances there are signs of pain manifested by frequent lying down, rolling, sighing, &c.; whilst in others the seat of pain is pointed at by the animal turning round and endeavouring to bite or scratch its loins. In one instance this symptom was very marked, and called forth the observation from the attendant that the horse wanted to bite its near hind leg, which was elevated and brought forward as if for that purpose; the animal, however, seemed to desire to bite its back, but was unable to do so. Should the suppression—*ischuria*—be prolonged, other symptoms, namely, those of blood poisoning—*uræmia*—arise. The retention of urea in the blood does not affect all animals alike. In the dog, insensibility and coma soon occur; but in the horse the effect seems to resemble a moderate alcoholic intoxication; the eye becomes brilliant, there is partial unconsciousness, but no loss of motor power. In one case, where total suppression continued for five days, no signs of coma or even somnolence were observed; but the animal became incapable of directing its movements, which were automatic, and from right to left, in which direction it continually moved round the box, and no force could compel it to move in the contrary direction. As the system becomes loaded with urea, the *feces* and secretion of the skin emit a strong uriniferous or even ammoniacal odour; the breath becomes *foetid*; the bowels constipated and tympanitic, with frequent vomition in the dog. In the case above mentioned, it was observed that the near (left) side was covered by a continual sweat, which became more and more uriniferous as the disease advanced; the skin of the opposite side was dry, hard, the coat staring, and on the fourth day the near hind limb was partially paralyzed; and the horse was frightened if it were suddenly touched or approached. In the stallion, retraction of the testicle on the affected side may be present.

*Post mortem appearances.*—In the acute form of general nephritis one or both kidneys may be involved; generally one is more especially affected, being enlarged, of a dark colour, and streaked with patches of congestion. It is easily lacerable, and if cut with the knife, a deep-coloured fluid flows from the cut surface, which, if examined microscopically, will be found to

contain pus cells in abundance; there is exudation into the tubes and desquamation of the epithelium, constituting "epithelial casts." Pyelitis, however, gives rise to the formation of abscesses, which are principally confined to the cortex of the kidney. These vary in size from a pin-head to a pigeon's egg, or even larger. Abscesses are multiple and surrounded by congested tissue, and sometimes by submucous hæmorrhage; this was specially observable in the cow referred to. They are easily seen through the capsule of the kidney as many yellowish white spots.

Interstitial nephritis consists of an inflammation of the interlobular connective tissue, similar to cirrhosis of the liver, and leads to contraction of the affected kidney.

*Treatment.*—This must be according to the ordinary principles of the treatment of inflammation. If the pulse be strong, a full bleeding will be of much service, not only allaying the febrile disturbance, but, by acting as an evacuant, removing much effete material from the economy. The intestinal canal is to be freely acted upon by cathartics, for direct experiment has shown that the urea, &c. which accumulate in the blood are by this means eliminated from the body, and the symptoms of uræmic poisoning delayed. For the horse, aloes; the ox, the sulphates of magnesia or soda; the dog, calomel and jalap, with enemata of warm water, warm fomentations or poultices to the loins, succeeded by mild mustard applications.

If the suppression continue for several days, or if at any time uræmic intoxication is apparent, it becomes necessary to excite the secretion of urine, and the best and safest method of doing this is by the application of digitalis to the skin in the form of a decoction, repeatedly applied as a fomentation to the loins, or as a poultice. It must, however, be discontinued immediately after the kidneys have commenced to react. I can speak with great confidence of this remedy if thus applied.

If pain be a prominent symptom it must be relieved by opium.

It is scarcely necessary for me to say anything against the application of cantharidine blisters, as the practitioner is aware that they have a most irritating effect upon the urinary organs.

As sequelæ to nephritis, atrophy and degeneration of the gland may result. If the inflammation be confined to one, it is

found that the other kidney generally becomes hypertrophied, having a double function to perform. In the case mentioned, where the suppression continued for five days, the left kidney was found to be, when examined some years afterwards, a mere flabby bag, its substance destroyed, and the ureter impervious; the right kidney, on the other hand, was much enlarged, and almost healthy. This condition of atrophy of one, and hypertrophy of the other, was diagnosed at the time it was attended, for the enlarged organ was easily detectable by the hand introduced into the rectum. Both Percivall and Gamgee quote a case of suppuration of the kidney, described by D'Arboval, occurring in a mare which had fallen into a hole.

#### ALBUMINURIA.

The occurrence of albumen may be due to various causes unconnected with disease of the kidneys, as in "red water" in cattle, some forms of indigestion in the horse, as well as from cerebro-spinal irritation. The application of large cantharidine blisters may also be followed by albuminous urine. According to some this is due to the toxic effects of cantharidine upon the blood, inducing a condition of that fluid simulating that resulting from the action of morbid poisons. According to others it is a result of the direct irritating effects upon the kidneys of the absorbed cantharidine. This latter view seems to me to be the more correct one, for not only does it irritate the kidneys, but the urinary passages generally, as manifested by frequent and difficult urination—*strangury*—which is best treated by opium, demulcents, as linseed tea, and bicarbonate of soda.

Persistent albuminuria arises from that degenerated condition of the kidney termed by medical writers "Bright's disease." It is, however, very rare in the lower animals. It is described by veterinary authors under the term of albuminous nephritis and granular degeneration of the kidneys.

The urine in this disease is permanently albuminous, and if examined microscopically will be found to contain a number of thread-like cylinders, which are in fact slender fibrinous coagula moulded to the shape, and discharged from the urinary tubes of the kidneys. They are generally studded with minute epithelial cells, which have been detached from the surface of the urini-

ferous tubes, and *in some instances* they are mixed with oil globules, which denote not only the presence of desquamative nephritis, but of fatty degeneration also.

The anatomical characters of the kidneys in this disease are of two kinds, and are spoken of as the *large white* and the *small red* kidney. If a longitudinal section of the large kidney be made, its cortical portion is seen to be much increased; the organ is soft in consistence, smooth upon its surface and upon its investing membrane; whilst the small kidney is hard and red, rough upon its surface, and its investing membrane firmly adherent.

*Symptoms.*—In addition to some stiffness of gait, there is continued desire on the part of the animal to stretch out in the stall, and in this position to continue, as described by Mr. Percivall, “with its fore legs extended under the manger, and its hind ones backwards, unless disturbed, all day long, not for the purpose of staling, but apparently because that posture seemed an easy or a comfortable one to it.” The diagnostic signs are present in the urine itself, which is albuminous, containing fibrinous casts of the uriniferous tubes, epithelial cells, or perhaps oil globules, whilst in quantity it may be very materially diminished or increased. I had the opportunity of watching the progress of a case for a number of years, and of making a *post mortem* examination after the animal’s death. The general health seemed but little affected. There was some shortness of breath, the animal never lying down, and some stiffness of gait; the urine varied in colour and density, sometimes dark, sometimes pale, but always albuminous and copious in quantity. The horse became paralyzed, and was destroyed. Examination of the kidneys showed that they were red and small, with numerous cysts, containing a thin transparent fluid in their interior. I was unable to account for the copious secretion of urine, but I find the same thing occurs in man; and that in the large white kidney the secretion is scanty, has a higher specific gravity, and contains clear, fibrinous, wax-like, and sometimes oily casts, with occasionally a little blood, and is frothy; whilst in the small red kidney the urine is copious as to quantity, and in the advanced stages pale, of a very low specific gravity, and contains granular casts of the uriniferous tubes.

*Treatment.*—When resulting from degenerative changes in the

glands, treatment of albuminous urine can only be palliative, relieving the kidneys as much as possible from the labour of elimination, by keeping the bowels in a relaxed condition by proper food, the skin warm, avoiding exposure to cold, and preventing anæmia by mineral tonics or the mineral acids, and putting the animal to such labour as it is capable of performing. When arising from other than disease of the kidney, albuminous urine, which is but a symptom, will disappear as the causes of such diseases are removed.

#### FLOATING KIDNEY.

I have met with one case where the right kidney in a cat was situated subcutaneously between the two last ribs. Supposed to be a tumour which had arisen in consequence of a bite from a dog, it was removed by me early in 1879; and, what is most interesting, the cat was none the worse of the operation, and is alive at the present time and as well as ever.

#### HÆMATURIA—RENAL CALCULI.

The occurrence of blood in the urine is due to a variety of circumstances, as acute congestion, degenerations already described, cancers, melanosis, and to the presence of calculi.

Renal calculi in the horse are composed of the carbonate of lime, and their presence is discoverable by the condition of the urine, which is charged with earthy materials, and by intermitting discharges of blood, occasional colicky pains, more especially after the animal has been severely worked or exercised. They are, however, exceedingly rare, and are best treated by the administration of hydrochloric acid, by the avoidance of food and water rich in saline matters, and by keeping the digestive and secretory functions well regulated by diet, careful grooming and exercise when admissible, and the avoidance of hard, calcareous water. In some instances the calculi become impacted in the ureters, inducing a more or less rapid degeneration of the kidney and suppuration within its substance, causing extreme agony and death. If the ureter becomes much dilated, the dilatation may be discovered by an examination *per rectum*, in the form of a fluctuating swelling upon either side of the pelvis.

## CHAPTER LXXX.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (S) CYSTITIS, OR INFLAMMATION OF THE BLADDER.

EXCEPT as a result of the irritating effects of cantharidine or croton oil absorbed from a blister, or administered internally, and of the application of turpentine, or too large a dose of it internally, I have never seen this affection in any of the lower animals, and am disposed to coincide with many veterinarians, that it never occurs except from the above causes.

*Symptoms.*—These are manifested primarily by excitement, followed by prostration of strength, the animal appearing to suffer much pain and distress; the urine is passed frequently, and with difficulty and pain; the fæces are covered with mucus and mixed with blood. When induced by cantharides or croton administered by the mouth, the inflammation extends throughout the whole alimentary canal, and there is redness of the buccal mucous membrane, with difficulty of swallowing, and attempts at vomition in horses and cattle, the genito-urinary organs being also much affected; but if the above irritants be absorbed from a blistered surface, their effect is concentrated upon the urino-genital organs, causing in most animals strangury and apparent sexual excitement, and if these symptoms are not soon allayed, death will result in two or three days. In turpentine poisoning the urine has a distinct odour of violets.

*Treatment.*—If from a blister, wash and cleanse the blistered part; internally, hyoscyamus or opium, bicarbonate of soda, demulcents, such as linseed tea, mucilaginous drinks, milk, and white of eggs will be found beneficial.

#### RETENTION OF URINE.

An inability, total or partial, of expelling by natural effort the urine contained in the bladder. It is caused by spasm of the



neck of the bladder; so induced, it is often a complication of colic; paralysis of the bladder. Retention is a common complication in parturient apoplexy and in paraplegia; enlargement of the prostate in aged males; cystic or urethral calculi; cancer of the penis; any cause of obstruction at the urethral opening; prolapsus of the uterus or vagina, and excessive accumulations of fæces in the rectum, and of dirt in the sheath. Inability to urinate may also result independently of the above causes and of any disease of the bladder or urethra, when an animal is unable or unwilling to rise upon its feet, as in azoturia, laminitis, or paralysis.

The symptoms are frequent and ineffectual attempts to urinate; if standing, the animal will stretch itself out, strain violently, and groan with pain, discharging but a few drops of urine, or none at all. Examination *per rectum* will enable the practitioner to feel the distended bladder with the hand, and this distension of the bladder is the diagnostic symptom. Such an examination will also often enable him to discover the cause; if from enlarged prostates, these will be felt as oval bodies immediately within the pelvis pressing upon the urethra. I have one case recorded where the animal had suffered, according to my informant, for five weeks from what appeared to be incontinence of urine—there being a continual dropping of urine night and day—but which proved, upon examination, to be a case of retention from the pressure of large prostates. The bladder was enormously distended, and the continual dropping of urine was the mere overflow; upon the catheter being introduced relief was immediately given by the withdrawal of a large quantity of urine. It was necessary to introduce the catheter two or three times a day for about a week, when the enlarged prostates, under the influence of iodine internally administered, and applied as an ointment to the perinæum, diminished in size, and permanent relief was afforded.

Paralysis of the bladder, if not caused by cerebro-spinal or spinal derangement, may be induced by retention of urine, as when an animal is compelled to perform a long journey without the opportunity of relieving itself. In this case the muscular fibres of the bladder, unnaturally stretched by the pressure of the contained urine, lose their tonicity, and become unable to contract upon their contents. Whatever be the cause of

the retention, relief must be afforded by the introduction of the catheter, and it is highly important, in all cases where animals retain the recumbent posture, to examine the condition of the bladder, and afford the necessary relief by the introduction of the catheter. Sometimes, however, the evacuation may be effected by firm but not violent pressure upon the bladder with the open hand introduced into the rectum, taking care that the mucous membrane of the rectum be not injured by the finger nails or by too violent pressure. In introducing the catheter in the cow, care must be taken not to injure the thin, delicate, membranous valve which guards the urethral opening; and in order to introduce the catheter without causing injury, the valve is to be lifted upwards by the finger, and the point of the catheter carefully introduced beneath it. If retention be due to the accumulation of dirt within the sheath of the penis, it may not be necessary to introduce the catheter, but to wash the parts thoroughly with soap and warm water; a little carbolic acid in the latter will render this operation much less offensive to the nostrils by destroying the foetid smell.

#### INCONTINENCE OF URINE.

This is the reverse of retention, being a continual flow of urine. It arises from a variety of causes, namely, paralysis of the sphincter vesicæ, the muscular power of the walls of the bladder remaining intact; calculi; and from pervious urachus shortly after birth.

For the treatment of the two latter causes see *Principles and Practice of Veterinary Surgery*; and for that arising from paralysis cathartics may be necessary, succeeded by nux vomica and cantharides, with injections of cold water into the rectum.

“Incontinence is said to occur in pigs after eating of polygonum, hydropiper, and lapathifolium.”—(GAMGEE.)

The following terms are applied to the checked discharges of urine:—suppression—*ischuria*; painful discharge of little urine—*dysuria*; and the passage of urine in drops—*strangury*.

## CHAPTER LXXXI.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

##### (T.) HYSTERIA.

CONCURRENT with the period of œstrum in the female, the following symptoms—clenching of the jaws, grinding the teeth, difficulty of swallowing, some degree of trismus, squinting of the eyes, tonic spasms, alternating with those of a clonic kind, continual kicking in an irregular uncontrolled manner with one or both hind feet, stamping of the fore feet, and other signs of nervous excitement, have been observed in the mare and female ass.

In one case that I saw, the two fore shoes and one hind shoe had been thrown off by the violent kicking and stamping. Attempts were made to remove the remaining one, which was loose; they were, however, unavailable, as the slightest touch caused a most violent kicking. The animal would fall, the whole trunk and neck being perfectly rigid, whilst the limbs continually moved in a violent and spasmodic manner. The power of volition seemed to be completely lost. The gluteal muscles were exceedingly hard and prominent. The rapid opening and closing of the vulva and its highly injected mucous membrane indicated the hyperasthæsia of the genital organs. Urine was passed abundantly, at first pale and watery, but afterwards highly coloured, and loaded with solid matters.

In another case the symptoms were rather different. The animal was prostrate, rigid, with its eyes turned upwards, as if in a trance. They were both bled, and an endeavour was made to nauseate them with aloes, and to allay the nervous irritability with opium. One recovered, the other died. For the prevention of this condition I can only suggest that the sexual desire be gratified if possible, whenever such a state of excitement short of the above presents itself.

## CHAPTER LXXXII.

### SPORADIC DISEASES—*continued.*

#### LOCAL DISEASES—*continued.*

#### (U.) DISEASES CONCURRENT WITH OR IMMEDIATELY SUCCEEDING PARTURITION.

ACCIDENTAL conditions connected with parturition, requiring the aid of a surgeon, will be found fully described in the *Principles and Practice of Veterinary Surgery*, as it is my intention to confine my present observations to those of a more purely medical character.

Under the common term “milk fever” at least three separate diseases are generally described, namely, parturient fever, parturient apoplexy, and acute metritis, already described.

#### PARTURIENT FEVER.

*Definition.*—A benign form of fever, seen in the cow, mare, &c., occurring about the second or third day after parturition, and generally terminating in recovery in from twenty-four to forty-eight hours.

The *symptoms* are those characteristic of general febrile disturbance; the pulse generally full and strong, the breathing accelerated, the visible mucous membranes injected, the secretion of milk suspended, the mammary gland hard and slightly erythematous, bowels constipated. The cow may assume the recumbent posture, but the power of maintaining the standing one is not lost, nor are there any signs of unconsciousness or of cerebral disturbance. Unlike parturient apoplexy, it attacks animals of all ages, but it is most commonly met with after the first parturition, and is induced by cold, or some dietetic error,

and seems to be connected with that condition of excitement associated with the secretion of milk.

*Treatment.*—The treatment of this affection is very simple, a mild oleaginous purgative, combined with a dose of the spirits of nitrous ether, warm clothing, and a restricted diet being all that is required.

#### ACUTE METRITIS, OR INFLAMMATION OF THE WOMB.

A very fatal form of inflammation, occurring in a few hours or within two or three days after parturition.

Whilst parturient apoplexy and parturient paralysis are generally met with in the cow, acute metritis, or, more correctly, metro-peritonitis, attacks the cow, mare, ewe, the bitch, and the sow.

*Pathology.*—A diffuse inflammation, primarily situated in the mucous membrane of the uterus, soon, however, extending to the uterine veins, giving rise to the formation of clots or thrombi, and extending to the peritoneum and intestines. The inflammation is characterised by its tendency to spread rapidly over a large surface, and by the rapid formation of an abundant quantity of a dark chocolate-coloured fluid exudation, which stains the tissues of the organs involved, and is poured out upon the free surface of the uterine mucous membrane, from whence it is discharged *per vaginam*, and which, by its acidity, causes much irritation and straining—tenesmus. Examined microscopically, it is found to consist of the *débris* of disintegrated blood globules, pus, and blood corpuscles, and an abundant quantity of granular material slightly intermixed with shreds of imperfect lymph floating in a reddish-coloured serosity.

This affection may be looked upon as being due to pyæmic or ichoræmic poisoning, the whole mass of blood becoming altered in its character, dark, feebly coagulable, with the appearance of secondary spots of inflammation and ecchymosis in the lungs, the brain, and other organs.

The occurrence of the disease is due to over-fatigue, as from over-driving immediately prior to the act of parturition, wounds inflicted upon the uterus or vagina during difficult delivery, the retention of the foetal membranes, which, rapidly decomposing, infect the blood by absorption of the putrescent products. It

may also be induced by obesity (more particularly in the bitch), exposure to cold, or any other debilitating influence.

Such predisposing causes are, as far as possible, always to be avoided, and the strictest hygienic conditions observed at this critical period. The organisms which set up metritis may be various, but the commonest are *B. coli*, staphylococci and streptococci. The latter are the most dangerous as so frequently tending to result in septicæmia. It will be seen, therefore, that every care should be taken in sterilising all instruments, hands, &c., when dealing with parturient cases. It is extraordinary what an amount of septic material may, in many cases, be introduced to a cow's womb without causing any trouble (see *Septicæmia and Parturient Fever*, p. 462); but this is no reason why the intelligent veterinarian should take any risks, and especially so when dealing with mares and bitches.

The disease may occur within a few hours after parturition, or its appearance may be protracted to the third or fourth day, and the chances of a favourable termination are in accordance with the lateness of the attack. If occurring within the shorter period, it is almost invariably fatal.

*Symptoms.*—Restlessness, paddling of the hind feet, loss of appetite, almost a total suppression of milk, the few drops that might be squeezed from the mammary gland being of a bluish tint, thin, and watery, sometimes of a reddish or even chocolate colour, containing granular matter, more or less curdled in appearance, and exhaling a peculiar odour. The vulva is small, contracted; the vaginal mucous membrane sometimes of a dark purple hue, or a deep red colour, with petechial spots and purple streaks. There is violent straining, discharge of a coffee-coloured, foetid liquid, and, as the disease advances, an exhaustive foetid diarrhoea. The prostration of strength is extreme; the pulse is feeble, thready, and very rapid; the heart's action tumultuous; the belly tympanitic; the surface of the body cold. So long as sufficient strength remains, the animal will frequently lie down and rise again; will kick the belly, and show other signs of abdominal pain; and in the mare these symptoms are sometimes complicated with those of inflammation of the feet. As the debility increases, the animal will be unable to maintain the standing posture, but whilst recumbent, will still manifest symptoms of

abdominal pain by rolling from side to side, looking round at the flank, and by violent attempts to regain its feet; the pulse becomes feebler and feebler; the breathing hurried and thoracic; at last the eye becomes amaurotic, and complete unconsciousness sets in; the animal dying in from twelve hours to perhaps two days after attack.

Whilst practising in Mold, I had the opportunity of seeing this disease extensively. Mold at that time was the terminal station of the railway, and calving cows were driven in from long distances in Wales for the purpose of being conveyed to the English markets. They would sometimes calve on the road, and were of necessity driven onwards with the rest of the herd, and too often succumbed to this fatal malady.

*Treatment.*—Bleeding, purgatives, and depressants of all kinds hasten the fatal termination, and the only hope of recovery must be based upon attempts to restore the diminished vital powers by the administration of alcoholic stimulants, the removal of the uterine and abdominal pain by sedatives and fomentations to the loins and abdominal walls, and to destroy the septic properties of the contents of the uterus, and soothe its irritated and inflamed mucous membrane by injections of warm water containing opium and antiseptics, such as "Condy's Fluid," hyposulphite of soda, or carbolic acid largely diluted. It must be remembered, however, that carbolic acid preparations must not be used in bitches, as they are extremely susceptible to poisoning by such preparations. It is now generally conceded that the use of antiseptics of an astringent nature is, as a general rule, to be avoided. Boiled sterile water should be used for douching and washing out internal cavities such as the uterus. Astringents coagulate the serous effusions, and so form a suitable culture medium for the many micro-organisms which lie in the deeper structures, and which are not killed by the application of the antiseptic. Moreover, the phagocytes are also killed, as easily as the organisms, by the antiseptic douche; thus a strong defence is broken down, and by the above-mentioned coagulation of the serous effusion diapidesis is impeded.

Amongst ewes the disease is known by the term "inflammation," and great success has been obtained in its treatment by the application to the inflamed uterus of carbolic acid one

part, olive oil ten parts. The same treatment is applicable to other animals.

*Contagious Mammitis*.—All mammites may be said to be contagious, inasmuch as they are all due to the infection of the gland by micro-organisms, which under certain conditions find a suitable nidus, and produce the usual symptoms of inflammation. There are numerous special organisms that can be cited, excluding the ones referred to below. Staphylococci, *B. coli*, streptococci, and diplococci are associated with such conditions.

Injuries to the udder may not result in microbic invasion, but in the majority of cases they do, as the damaged tissue loses its resistance, and organisms find their way in. Specific diffuse tubercular mammitis may be diagnosed roughly by its slowly progressive character, and the fact that the affected quarter tends to progressively increase in size and become harder and larger than its fellows; whereas in other inflammatory conditions, known as garget, after the first acute inflammatory symptoms have subsided, the functional activity is so reduced that the quarter wastes, and usually becomes smaller than its fellows. To justify a diagnosis of tubercle in the udder it is essential to find the organisms by inoculation of guinea-pigs (*vide* tubercle), and so differentiate it from the somewhat similar lesions caused by actinomycosis.

Another specific form of contagious mammitis is that which now prevails very largely at home, in our Colonies, and in many foreign countries, and sometimes rages as an enzootic, or even assumes an epizootic, type. It is stated by Nocard and Mollereau to be due to a rounded or ovoid micrococcus  $1.25\ \mu$  in length by  $1\ \mu$  in thickness, and forming long, straight or sinuous chains, sometimes bilobed by way of division, and found in the milk and the walls of the excretory ducts. It is aërobic and anaërobic, and its growth in cultures is arrested by weak solutions of boracic acid, and by a 3 per cent. solution of carbolic acid, and it is recommended that the milkers of cows thus affected should wash their hands, previous to milking, with this carbolic solution. Unless the disease be checked at its onset, there is a great probability of it assuming a malignant type, and terminating in gangrene and death of the patient.



Experimental inoculations with pure cultures into the teat have reproduced the disease in the cow and goat. Even on the first day the milk from the inoculated udders was swarming with micrococci, had an acid reaction and a curdled appearance, and finally the udder became inflamed. A fatal form of gangrenous mammitis is seen in milch ewes—said by Nocard to be due to a micrococcus  $0.24\ \mu$  in diameter, associated in groups of four or more, but never in chains, stainable by Gram's method, anaërobic, coagulating the milk and turning it sour. Curdling of milk, as well as other changes—viz., putrefaction, viscous milk, blue milk, red milk, and yellow milk—are all due to various microbes, and should be counteracted by cleanliness and the application of antiseptics, such as boracic acid.

## CHAPTER LXXXIII.

### PARASITIC DISEASES.

REMARKS ON CLASSIFICATION—NEMATODA—TREMATODA—CESTODA—ACANTHOCEPHALA—DIPTERA—TRACHEARIA—TABLES OF ENTOZOA.

#### CLASSIFICATION OF PARASITES.

PARASITES are distinguished as *Endoparasites* when living in the interior of their hosts, and as *Ectoparasites* when external. With the *Ectoparasites* we have at present nothing to do, but will confine our attention entirely to the *Endoparasites*. Among the most important endoparasitic animals or Entozoa are those spoken of collectively as "worms," including not only round-worms resembling the earth-worms (*Lumbricus terrestris*), and hence popularly spoken of as *Lumbricoids*, but also worms resembling a band or a tape (tape-worms), or a leaf (flukes), as well as the thorny-headed worms (*Echinorhynchus*). The round-worms and thorn-headed worms form together the class *Nemathelminthes*, while the flukes and the tape-worms constitute the class *Platyhelminthes*. These two classes form the parasitic division of Cuvier's sub-kingdom VERMES.

These classes are further broken up into natural orders; thus the class *Nemathelminthes* includes the two orders *Nematoda* and *Acanthocephala*, and the class *Platyhelminthes* the two orders *Trematoda* or flukes, and *Cestoda* or tape-worms.

These natural orders are further subdivided into families, genera, and species. Then, again, it must not be forgotten that there are other kinds of internal parasites, commonly designated "bots." These creatures are not usually classed with the entozoa, or helminths proper, because they are merely the larval stages

of growth of various species of gadfly. These flies are generally included in the genus *Æstrus* of entomologists, the *Æstridæ* forming a rather numerous family of the class INSECTA, and belonging to the order called DIPTERA. As the attention of the profession is often called to these singular creatures, it will be desirable to give a brief account of their development and habits.

There is yet another series of internal parasites, long ago called Pentastomes, from the notion that they were furnished with five mouths. These worms are also occasionally brought under the notice of the practitioner of veterinary medicine; and in one remarkable instance recorded by the late Professor Dick, three of these entozoa caused the death of a valuable sporting dog. These Pentastomes, in fact, are allied to the true ticks, and therefore must be grouped along with all those spider-like creatures, which, in common with the spiders themselves, breathe by means of peculiar air-vessels termed tracheæ. In other words, the Pentastomes belong to the order TRACHEARIA, forming a division of the class ARACHNIDA.

It thus appears that, without taking into consideration certain minute parasitic organisms belonging to the lowermost class of animals, and misnamed "cattle plague bodies," we have to deal with no less than six well-marked orders of invertebrate animals, one or more species of each of these orders being liable to play the part of parasite within the body of some one or other of our various domesticated animals.

The pathogenic role played by these parasites has not hitherto received the attention that it deserves. The amount of disease indirectly set up by these intestinal parasites is enormous. The special study of intestinal parasites has, until quite recently, been treated as being more or less superfluous, but each day tends to dispel such an erroneous notion.

It is not that these parasites in themselves do very much harm, but it is the predisposition to harm which they incite. The point of attachment of even a bot, not to mention the grosser lesions caused by the larger intestinal worms, becomes to all intents and purposes a wound, and such wound may be the means of bacterial organisms being introduced to the animal's economy. The fight for existence against pathogenic organisms is already very intense, and the aid to these organisms which parasites of the larger animal type give is considerable.

Giles and Baldrey in India have pointed out that some of the ravaging outbreaks amongst sheep are in all probability induced by these means. An animal's strength is sapped, and resistance to disease lowered, by the constant drain of nourishment for the large numbers of intestinal parasites, so that at the first onslaught, by comparatively harmless pathogenic organisms, the animal succumbs.

We all know to what an extent kennels are sometimes decimated by the persistency amongst puppies of attacks by intestinal parasites, and how such infected animals are the first to succumb to distemper, eczema, and pneumonia.

The subject of parasitism is, therefore, one worth considerably more attention on the part of the practitioner than it has had hitherto. Treatment in this field has mostly been left to the quack and retailer of patent nostrums, but the time has arrived when the order must be changed.

To such of the parasites as have an especial interest for the profession, and which are included in one or other of the six orders, namely, NEMATODA, TREMATODA, CESTODA, ACANTHOCEPHALA, DIPTERA, and TRACHEARIA, the reader's attention is now invited.

## CLASSIFICATION OF ENTOZOA.

### SUB-KINGDOM VERMES (WORMS).

#### Cl. I. *Platyhelminthes* (Flat-worms).

(N.O. 1.) *Cestoda*.

(Fam. a.) *Tæniadæ* (Tape-worms).

Gen. *Tænia*.

(Fam. b.) *Bothriocephalidæ* (Pit-headed worms).

Gen. *Bothriocephalus*.

(N.O. 2.) *Trematoda* (Flukes).

Gen. *Distoma*.

*Schistosoma* or *Bilharzia*.

*Amphistoma*.

#### Cl. II. *Nemathelminthes* (Round-worms).

(N.O. 1.) *Nematoda* (Round-worms proper).

(Group a.) *Polymyaria*.

Gen. *Ascaris*.

*Eustrongylus*.

*Filaria*.

*Spiroptera*.

- (Group b.) *Meromyaria*.  
                   Gen. *Oxyuris*.  
                           *Strongylus*.  
                           *Ankylostoma* or *Dochmius*.  
 (Group c.) *Holomyaria*.  
                   Gen. *Trichina*.  
                           *Trichocephalus*.  
 (N.O. 2.) *Acanthocephala* (Thorn-headed worms).  
                   Gen. *Echinorrhynchus*.

## SUB-KINGDOM ARTHROPODA.

- Cl. I. *Insecta*.  
           N.O. *Diptera*.  
                   Fam. *Cestridæ*.  
                           Gen. *Cestrus*; the larvæ of some species are  
   Entozoa.  
 Cl. II. *Arachnida*.  
           N.O. *Trachearia*.  
                   Gen. *Pentastoma*.

## NEMATODA.

This order is treated of *first* because it includes the parasites emphatically called "worms." When a horse is said to have worms, we know that in nine cases out of ten the animal is passing lumbricoids of large size (*Ascaris megalocephala*), or it is troubled with maw-worms (*Oxyuris curvula*). In like manner, when a dog is said to have worms, it is either meant that the animal is passing or throwing up round-worms (*Ascaris marginata*), or that it is parting with the falsely so-called maw-worms, which, after all, are neither more nor less than the free and independent segments or proglottides of some species of tape-worm. Worms in cattle and sheep are not often spoken of as such, and little attention is paid to those of the pig. With the cat, however, it is far otherwise, for we have known veterinarians whose assistance has been eagerly sought in view of ridding this domestic pet of its internal enemies, and especially of the nematode entozoon called *Ascaris mystax*. In addition to the above, there are other species belonging to the nematode order, to which a larger interest is, or ought to be, attached by the

professional man. To this series belong the little flesh-worm (*Trichina spiralis*); the thread-worm which occasions the "lamb disease" (*Strongylus filaria*); the very similar nematode that produces husk or hoose in cattle (*Strongylus micrurus*); and the worm which gives rise to aneurisms in the horse and ass (*Strongylus armatus*). No member of the veterinary profession should be totally unacquainted with the natural history of these important species of parasite.

The *Strongylus filaria* of sheep and calves causes parasitic bronchitis from its presence in the bronchial tubes. The development of these parasites is comparatively simple, the mature worms are ovoviviparous and the embryos are expelled on the pastures, where they moult, further develop, and live for some time, until again taken up by cattle or sheep to complete their evolution. They are taken in either with wet grass or water, and from the stomach find their way to the lungs.

The *Strongylus rufescens*, a very minute worm, is found in the lung tissue of sheep, and often causes a pneumonia. They may become encysted and set up a tubercular condition which has been called pseudo-tuberculosis. The eggs are hatched in the mucus of the bronchial tubes and their terminals, and the embryos are expelled with the mucus therefrom. They can live a long time in damp places, and whilst there, moult and prepare for invasion of their special host.

We have not an entire knowledge of the course of development undergone by all of the above-mentioned nematodes. It is true that, with more or less deviation from a common plan, all of them must pass through similar stages of growth, from the time of their first formation in the egg up to the period of sexual maturity. All, moreover, must in some way or other conform to a known law of their existence, which involves in many species at least one change of residence and skin before they can acquire the adult condition within the body of the last or ultimate bearer. Herein lies the difficulty in tracing out the development of most of the species; nevertheless, from the facts already made known by experimental research, it is not too much to hope that we shall hereafter become possessed of a knowledge of all the more important phenomena connected with the development of the nematode worms.

The *Trichina spiralis*, as ordinarily known, is a small sexually

immature nematode, usually found lodged within capsules or cysts, and occupying the muscles of some animal, such as the rat, the pig, or of man himself. When the little worm is removed from the cyst, its entire length will be found not to exceed the  $\frac{1}{25}$ th of an inch. In its full-grown or sexually mature state it is still a very minute worm; the males measuring only the  $\frac{1}{18}$ th of an inch, whilst the females, which are more than as large again, reach to about  $\frac{1}{8}$ th of an inch.

The professional importance of the trichina more directly concerns the medical man than the veterinarian; nevertheless, since the human disorder termed trichiniosis takes its origin from the consumption of animal food, especially pork, it is clearly the duty of the latter to understand the nature of the malady thus provoked, and to acquaint himself with the phenomena of the parasite's development. The experiments of various helminthologists, and especially those of Davaine, have distinctly proved that when small animals, such as rats, rabbits, and cats, are largely infected, they, like man himself, readily succumb to the disease. In the case of larger animals, a very great amount of infection is necessary to give rise to any external symptoms. So complete, indeed, does this immunity appear to be, that a pig experimented on at the Royal Veterinary College showed no sign of the disorder, although from subsequent *post mortem* evidences it was calculated that its flesh contained sixteen millions of living worms.

Until very lately we flattered ourselves that in England and Scotland there was no such thing as trichina existing in our home-reared porkers; but so far as the former division of the country is concerned, this immunity can no longer be said to exist. Not only have several English fed pigs been found to harbour spiral flesh-worms, but in the year 1871,\* as recorded by Dr. Dickenson, an outbreak of trichiniasis occurred in a farmer's family in Cumberland, this attack resulting from the consumption of pork reared by themselves. As Mr. Gamgee has well remarked, "If pigs are permitted to swallow the germs of human parasites, as in Ireland and in many British piggeries, we must expect hams, bacon, and pork sausages to be charged with the embryonic forms of human entozoa." Very much more, of course, might be said on this subject in relation to questions

\* And as lately as this year, 1909.

of hygiene; but our object in these pages is merely to show the necessity of a general acquaintance with the subject.

The symptoms, whether occurring in man or animals, are generally believed to be due to the wounds and consequent irritation set up by the worms during their wanderings in the tissues of the host; but whilst this is true as a cause of the phenomena occurring in the second stage of the disease, it is obvious that the earliest symptoms, often accompanied with diarrhœa, are due to intestinal irritation alone. Some authors have, indeed, contended that there is no such thing as traumatic injury caused by the wandering parasites, but our best experimentalists in helminthology are one and all in favour of the view here advocated.

Whilst many admirable memoirs have been written on the structure and development of trichina (the literature of the subject being of very great extent), we have on the whole satisfied ourselves that the clearly enunciated statements and conclusions of Leuckart are worthy of every confidence. The following is a brief *résumé* of his conclusions, given almost in his own words:—

1. *Trichina spiralis* is the juvenile state of a little round-worm.
2. The mature *Trichina* inhabits the intestinal canal of numerous warm-blooded animals, especially mammals.
3. The intestinal *Trichinæ* attain sexual maturity on the second day after their introduction into the stomach.
4. The eggs are developed within the parent worm into minute filaria-like embryos, which are born free from the sixth day onwards.
5. The new-born young soon commence wandering, penetrating the intestinal walls, and passing directly through the abdominal cavity into the muscles of the host.
6. The directions in which they proceed are in the course of the intermuscular connective tissues, the majority of the embryos resting in the muscles of the abdomen and thorax.
7. The embryos penetrate the separate muscular bundles, and at the expiration of fourteen days they will have acquired the size and organization of the spiral flesh-worm.
8. Soon after the intrusion of the parasite the infested muscular fibre loses its original structure; and after a



while the spot occupied by the rolled-up entozoa becomes spindle-shaped, within which the well-known lemon-shaped cysts are formed.

9. The further development of the muscle trichinæ is altogether independent of the formation of these cysts, the walls of which become hardened by calcareous deposition, and thus, moreover, males and females are already distinguishable in the larval state.
10. The immigration of the young parasites in large numbers produces very serious or even fatal consequences; and thus it happens, that in proportion to the quantity of imported parasites the symptoms resulting will be either severe, dangerous, or even fatal.

It has been stated by some writers that even pigs occasionally display symptoms of trichiniasis, the signs of the disease in the animal being loss of appetite, quiescence, aversion to all kinds of movement, and even partial paralysis of the limbs. In the human subject it is well known that the symptoms are much more severe, many of the patients enduring the most intolerable agony, resembling acute rheumatism, until at length death mercifully comes to put an end to their sufferings.

Here it will not be out of place to mention that cats are liable to be affected with another disease very closely resembling trichiniasis, which may appropriately be called *olulaniasis*. The disorder is occasioned by a minute trichina-like nematode termed by Leuckart *Olulanus tricuspis*. It gains access to the lungs in the larval state, producing death by suffocation.

Our knowledge of the history of the development of the larger round-worms is very incomplete; nevertheless the causes of the prevalence of these worms in particular localities and during certain seasons are not far to seek. It is clear that their final stage of growth is accomplished with great rapidity, otherwise we should not meet with lumbricoids in pigs and puppies scarcely three weeks old. Large round-worms have also been found in very young colts. The ordinary lumbricoids of the horse, of the pig, and of man, so very closely resemble each other, that by some they are regarded as mere varieties of one species (*Ascaris megaloccephala*, *A. suilla*, and *A. lumbricoides*). Whether they are so or not is of little practical moment, for it seems quite

certain that a perfect knowledge of the earlier stages of development of any one of them would furnish a clue as to what obtains in the others. It is probable also that the lumbricoids of the dog and cat undergo similar changes.

The eggs of the common round-worm have been kept alive by Davaine for more than five years; and various observers have watched their development in fresh water up to the stage of imperfectly developed embryos, and have kept them alive in this condition for three months.

Dr. Davaine administered some of his five-year-old embryos to rats, and had the satisfaction of finding a few of their eggs in the fæces, with their embryos still living, and striving to get out of the shells. He administered others to a cow, also introducing some into the stomachs of dogs in small linen-covered flasks. As a general result, it may be said that the embryos escaped from their shell; but the contents of those eggs in which the process of yolk-segmentation had not arrived at the stage of embryonal formation remained undigested. So far back as the year 1853 Verloren reared embryos in the eggs of the dog's round-worm within a period of fifteen days in distilled water. Dr. Cobbold has also reared the embryos of this species (*Ascaris marginata*) in fresh water, and has kept them alive for a period of seventeen months. At the expiration of this period, and during the warm weather, some of them escaped from their shells.

According to Davaine, the eggs of many nematodes will readily retain their vitality though long exposed to dryness, but their contents will not go on developing during this period of exposure. In the case of *Ascaris tetraptera* of the mouse, however, embryonal formation goes on in spite of the absence of external moisture. He has noticed the same thing in the oxyurides of rodents. Dryness does not even destroy the eggs of *Ascaris lumbricoides* and *Trichocephalus dispar*. It would seem, in short, that the eggs of nematodes, which normally take up their residence in cats, dogs, and carnivorous animals which reside in arid regions, will develop embryos *in ovo* without a trace of moisture. Davaine is of opinion that it is not necessary that nematode embryos should pass through the body of any intermediary bearer; and he believes that they are often directly transferred to the stomach of their appropriate hosts whilst adhering in the condition of an impalpable dust to the coats of their bearers,

whence they are detached by the animals themselves when licking the fur. With the eggs of *Ascaris megalcephala* Dr. Cobbold has performed several experiments, having reared the embryos in simple fresh water, and found them capable of escaping from their shells during warm weather. He also succeeded in rearing these larvæ in pond mud, noticing at the same time that after their exclusion they grew more or less rapidly up to a certain point, after which they appeared to stop, as if waiting transference to some host for the further accomplishment of their larval growth. The addition of horse-dung to the soft wet mud in one case, and of cow-dung in another, neither appeared to advance nor retard the process of embryonal growth so long as the embryos remained in their shells. On the other hand, when the embryos were reared in simple horse-dung, purposely kept moist, they attained a higher degree of organization than did those which were reared in water and wet mud. Having watched hundreds of these larvæ under varying conditions, Cobbold came to the conclusion that after their escape from the egg, their growth, strength, and activity are favoured, if they happen to have gained access to fluid media containing impurities. Ditch or muddy pond water would appear to be eminently favourable to the development of the escaped larvæ up to a certain stage of growth.

That warmth is eminently favourable to the development of all kinds of parasites is a well-established truth; and in the case of most nematodes it appears to be absolutely essential to the formation and hatching of the embryos. Take the case of *Oxyuris*, for example. As Leuckart observes (*Die Menschlichen Parasiten*, Bd. ii., s. 326), "One only needs to expose the eggs of the human thread-worm to the action of the sun's rays in a moistened paper envelope, when in five or six hours the tadpole-shaped embryos become slender elongated worms, which are not unlike the sexually mature oxyurides in form, exhibiting rather lively movements under the influence of the warmth." The power of warmth is thus very obvious in the case of *oxyuris*, since without a certain degree of temperature the earliest embryonal change cannot be accomplished. As in the *oxyuris* of man, these early changes are sometimes accomplished whilst the eggs, discharged from the maternal worm, still lie in the fæces or rectum of the bearer; so also it is probable that similar

changes occur in the eggs of *Oxyuris curvula* whilst they still remain in the rectum of the horse. According to Leuckart, the escape of the embryos of the human oxyuris ordinarily takes place when the eggs are swallowed by a new human bearer; but from the observations of Heller, it is also quite certain that any person may infest himself by swallowing the eggs which have come from oxyurides dwelling in his own person. In either case the escape of the embryos from the egg is brought about by the action of the gastric juice acting upon the egg-shell. The further changes resulting in the formation of the perfectly mature oxyuris are accomplished within the alimentary canal of the bearer. Here we have cases in which the adult sexual form of the parasite lays eggs in the alimentary canal of its host. Favoured by the warmth and moisture of the alimentary canal, the embryo reaches a certain stage of development while still enclosed within its egg-shell. If these eggs, expelled with the fæces, reach a new host, the embryo is hatched, and develops into a sexual adult. This is the mode of development in *Trichocephalus affinis*, and almost certainly in *Oxyuris vermicularis* of man, *Oxyuris curvula* of the horse, and *Ascaris lumbricoides*.

Thus Dr. Heller found, *post mortem*, young round-worms, of the species *Ascaris lumbricoides*, in the intestines of an imbecile. There were eighteen specimens, the largest of which had only acquired the length of about half an inch, whilst the smallest gave a long diameter of only 2.75 millimètres, or let us say roughly the ninth part of an inch. Thus Heller, in part at least, bridged over the gap which had formerly existed between the size of the embryo at the time of expulsion from the egg and the large-sized ones which alone had been formerly observed in the alimentary canal, and in this case we are almost certain that the eggs are hatched in the alimentary canal, where the embryos attain sexual maturity.

The mode of development is usually slightly different in *Ascaris* and *Strongylus*, where the eggs have a thin shell, and the embryo enjoys for a time a non-parasitic existence in water or mud. Here it grows in size, but develops no sexual organs. Sometimes it accidentally attaches itself as a parasite to a fresh-water mollusc, but it undergoes no change there. Eventually, gaining access to a proper host, it reaches sexual maturity, and the cycle of its life is repeated.

The researches of Looss in Egypt have shown that *dochmius* of man—or, as it is now more commonly called, *ankylostoma*—gains entrance to the intestine in an extraordinary way. The larval forms are hatched in water and from there gain access to the bare human skin, a naked foot or hand, the armed larvæ rapidly penetrate the unbroken skin, leaving only a slight reddened spot, they remain in the subcutaneous tissue for a time, and then, by an instinct or natural law, find their way through the blood-stream or lymphatics to the lungs; from there they bore their way into the intestine, where they attain sexual maturity and set up the well-known symptoms called *Ankylostomiasis* or *Miners' disease*. Infection is probably the same in the domesticated animals, where, as Baldrey and others have shown, in India considerable anæmia is set up in sheep and dogs infested with these parasites.

It is possible also that some embryos are swallowed in drinking water, and thus gain direct entrance to the intestine. It is these parasites that are so often the cause of serious attacks of pernicious anæmia in packs of hounds. The conditions under which they live facilitate contagion, and the infection is often of a very fatal nature.

In the case of some of the strongyles, there can be no doubt that the earlier larval transformations are undergone after the eggs have been expelled and lodged within soft soil or mud. Leuckart has proved this in the case of *Strongylus hypostomus*, whose rhabditiform young cast their first skin in about three weeks, at which time, as happens with many other larval nematodes, they part with their tails.

The case is still more complicated when the nematode requires two hosts to complete its metamorphosis. In some cases it is the egg which is taken into the first or intermediate host, in other cases it is the larva. We have a good example of a parasite requiring two hosts in the case of the *Spiroptera obtusa* of the mouse. The eggs pass out of the alimentary canal of the mouse, and are eaten by the meal-worm. The eggs are hatched, and the larva, after living in the meal-worm for about five weeks, forms round itself a capsule of connective tissue. If meal-worms containing these encysted larvæ are eaten by mice, the larvæ leave their capsules and become the sexually mature *Spiroptera obtusa* of the mouse. The *Filaria sanguinis*

*hominis*, or *Filaria Bancrofti*, is another good example of a parasite which requires two hosts. The sexually mature worm is found in the human tissues, and is viviparous, producing numerous larvæ, which make their way into the blood. The blood is sucked by mosquitoes, and thus the larvæ get into the mosquito, where they increase in size, and undergo various changes. The larval filaria lose their sheaths in the stomach of the mosquito, penetrate the abdominal wall, and arrive finally at the proboscis of the mosquito, where they wait until the warm-blooded host is bitten, to which they gain entrance, and find their way to the lymphatics; there they become sexually mature, the females become gravid, and so the cycle is completed.

The researches of Roë have shown that the filaria known as *Filaria immitis*, is propagated by both *Anopheles* and *Culex* mosquitoes. The adult filaria live in the heart of the dog, numerous embryos are expelled by the mature females and taken up by the mosquitoes from the peripheral blood, and their cycle continued as explained above. Lingard has endeavoured to show that the disease known as Bursattee is due to a filaria which sets up the irritation resulting in the formation of the chronic indurated calcareous ulcerations characteristic of this infection, although the results of his experiments hardly appear conclusive. Lingard and others in India, Tuck and Ford in the Straits Settlements, have shown the frequent presence of mature filaria attached to the endothelium of the aorta in cattle and horses, where they excite a nodular condition, but no demonstrable pathogenic symptoms, unless cases of anæmia from no ascribable cause may be taken as such infection. The *Filaria papillosa* of the horse is apparently harmless when in the peritoneum, but dangerous when in the eye, from the inflammatory condition it sets up in that organ.

Pease has shown that embryonic filaria are responsible for setting up a condition of cedematous swellings of the skin, resembling secondary dourine.

In all these cases, except the eye-worm, their presence can, as a rule, only be diagnosed by the finding of embryos in the circulation, and, as in the case of man, these embryos may be only demonstrable in the peripheral circulation at night, at

which time mosquitoes bite. The *Filaria hæmorrhagica*, causing the so-called bloody sweat of horses in Hungary, and the filaria causing summer sores, may be found by a superficial examination of the diseased part of the skin, which will show the embryos.

It will be seen, therefore, that filariasis is by no means uncommon in cattle and horses, but its pathogenicity is at present but ill-understood, and the difficulties of showing to what extent it exists, and the amount of demonstrable disease set up, are very considerable.

Several cases are on record of the *Filaria Medinensis* affecting dogs and other animals. The life-history of this filaria differs from others in having for its host the fresh-water cyclops known as *Cyclops quadricornis*. The larvæ are extruded from the mature female and gain entrance to fresh water; here they gain entrance to the cyclops, where they go through various metamorphoses, and eventually become ingested into a fresh host by means of the drinking water. The embryos, as a rule, find their way to the subcutaneous connective tissue, usually at one of the extremities; here the mature female becomes gravid and increases very much in size. The irritation so produced causes a small ulceration, which by stimulating with cold water, cause the female to extrude embryos, and gain entrance to water, where the cycle is again carried on. It is probable that the species affecting animals are different to those affecting man, but the symptoms to which they give rise are the same. The first thing noticed is a small punctured hole, through which the head of the *Dracunculus Medinensis* can be seen. The method of removing it is by very careful and persistent traction; if the worm be broken off, considerable trouble is likely to arise, as its total length may exceed several feet. Natives in various parts of the East who are affected with this worm twist it round a piece of stick, gradually withdrawing it, an operation which may take days or even weeks.

A third example of a nematode requiring two hosts is the *Trichina spiralis*. The sexually mature worm lives in the alimentary canal, and is viviparous, producing numerous larvæ, which make their way out of the alimentary canal into the muscles, where they encyst. When flesh containing the sexually immature larvæ is eaten, the cystic stage comes to a

close, and sexual maturity is attained in the alimentary canal, where the larvæ are again produced.

The eminent Russian traveller Fedtschenko observed the primary larval changes to take place in the young of the filaria known as the guinea-worm during their sojourn in the alimentary canal of *Cyclopes*, these entomostracous crustaceans being destined to play the part of intermediary bearers. In like manner, there can be little doubt that the young of the strongyles, which occasion husk and lamb disease, undergo their primary changes of development either within soft mud alone, or within the bodies of small slugs and other minute denizens of herbage, or possibly within the bodies of larval insects and minute entomostracans inhabiting ponds, ditches, and running streams. It thus seems that whilst some nematodes can accomplish their developmental processes without any lengthened sojourn outside their final bearer, others, on the contrary, require particular, varied, and prolonged conditions which shall enable them to undergo certain preliminary changes altogether exterior to and apart from the bodies of their ultimate hosts. In short, as Leuckart points out, we have two distinct groups of strongyles: those which lead a free life in their larval state, undergoing a certain grade of development in mud and water; and those which pass through certain larval changes of growth within the bodies of insects and other intermediary bearers.

So much for the natural history of the nematoda, or order of thread-worms and round-worms, on which much more might be said, were we not limited to a general view of all the groups of internal parasites. As it is, the reader will not fail to perceive that, speaking generally, it is now clearly understood how cattle and sheep and other animals obtain one frequent form of lung disease. It is almost needless to add that the labours of helminthologists have thus contributed largely towards the formation of rational principles on which to base successfully both a radical and prophylactic method of treatment.

#### PLATYHELMINTHES.

The flukes and tape-worms which belong to this class have flattened bodies, hence the name *Platyhelminthes* or flat-worms. They are hermaphrodite, and are usually furnished with organs



of attachment, such as suckers and hooks. By being hermaphrodite it is understood that each segment of the tape-worm has both male and female organs, but each segment or zöoid does not impregnate itself. The spermatozoids of one segment gain entrance through the genital openings to another segment of either the same individual or another worm, and the same is the case with the flukes or flat-worms. Usually there is an alternation of generations, *i.e.*, the young one is not like its parent, but must pass through various metamorphoses in order to reach the adult form.

## TREMATODA.

This order comprises the flukes ; and it is of great interest to the veterinarian, inasmuch as he is occasionally consulted in reference to the well-known disease in sheep termed *rot*. This disorder is unquestionably due to the presence of the common liver fluke (*Distoma hepaticum*), a parasite that is seldom more than an inch in length. Other animals than sheep are liable to be infested by it, but, except in the case of cattle, hares, and rabbits, it only very rarely occasions severe disease. In about a score of instances a similar entozoon has been detected in the human body. Similar ones also infest the horse.

For many years past investigations have been conducted with the special purpose of ascertaining the manner in which cattle and sheep infest themselves with this parasite ; we have succeeded in tracing out all the stages of growth of the common fluke, and researches amongst the trematodes generally have enabled observers to arrive at conclusions of the highest practical importance. Intelligent cattle-breeders and agriculturists have all along observed that *rot* was particularly virulent after long-continued wet weather, and more especially so when there had been a succession of such seasons ; and further, that the flocks grazing in low pastures and marshy districts were much more liable to invasion than those which pastured on higher and drier grounds ; but, what is most interesting, they also observed that an exception occurred in the case of those sheep feeding in the extensive salt-water marshes bordering our eastern shores. It was probably this latter circumstance which suggested the common and useful practice of mixing salt with the food of sheep and cattle,

both as a preventive and curative agent. At all events, as will appear in the sequel, the intelligible explanation of the good effected by this simple practice, is intimately associated with a correct understanding of the mode of development of the parasite in question.

The symptoms, treatment, and pathological appearances connected with *rot* are not here discussed, the present chapter being limited to the natural history of the entozoa. Even within this limitation the subject is too large to be treated of exhaustively and in detail; but for all practical purposes it is probably sufficient to follow the plan we have adopted in the case of trichina, namely, to offer a series of conclusions, such as appear to be well established by the independent researches of various helminthologists. In this connection it is only necessary to add that the labours of Steenstrup, Leuckart, Van Beneden, Pagenstecher, Moulinie, Davaine, Filippi, and Simonds have played a most conspicuous part.

1. The liver fluke, in its sexually mature state (*Distoma hepaticum*) gives rise to the disease commonly called *rot*; this affection being also locally termed *coathe* (Dorsetshire, Devon), *iles* (Cornwall) and *bane* (Somersetshire). In France it is known as the *Cachexie aqueuse*, and more popularly as *pourriture*. In Germany the epidemic disease is called *egelseuche*, and in a more limited sense either *die Fäule* or *die Leberkrankheit*.
2. The *rot* is especially prevalent during the spring of the year, at which time the fluke itself, and innumerable multitudes of the free eggs, are constantly escaping from the alimentary canal of the bearer. The impregnated eggs are thus ordinarily transferred to open pasture-grounds, along with the fæces of the bearer.
3. As it has been shown by dissections that the liver of a single sheep may harbour several hundred flukes, and as also a single adult fluke is capable of throwing off several thousand eggs, it is certain that any *rot*-affected flock is capable of distributing millions of fluke germs in the egg condition.

4. Such flukes as have accidentally escaped their host *per anum* do not exhibit powers of locomotion sufficient to enable them to undertake migrations. Their slight movements, however, subserve the purpose of concealing them in the grass where they have fallen, and also, probably, aid in the further expulsion of eggs, which latter can only pass from the oviduct in single file, one at a time.
5. After the death of the escaped flukes, the further dispersion of the eggs is facilitated by the subsequent decomposition of the parent worm, and also by its disintegration, occasioned by the attacks of insects. It has been calculated that the uterus of a full-grown fluke may contain upwards of forty thousand eggs.
6. By the agency of winds, rains, insects, the feet of cattle, dogs, rabbits, and other animals, as well as by man himself, the freed ova are dispersed and carried to considerable distances; and thus it is that a considerable proportion of them ultimately find their way into ponds, ditches, canals, pools of all kinds, lakes and running streams.
7. The eggs at the time of their expulsion exhibit the already segmented yolk in a state of fine division. The egg contents continue to develop outside the parent's body; the granular matrix finally becoming transformed into a ciliated embryo, which, when set free, follows the habit of infusorial animalcules in general by swimming rapidly in the water. The escape of the embryo is effected at the anterior pole of the egg-shell, which is furnished with a lid that opens in consequence of the action of prolonged immersion, aided by the vigorous movements of the contained embryo.
8. The ciliated, free, swimming embryo of the common fluke, at the time of its birth, exhibits the figure of an inverted cone, its anterior extremity, which is broad and somewhat flattened, supporting a central proboscis-like papilla.

A small pigment spot, placed dorsally, and having the form of a cross, is supposed to be a rudimentary organ of vision. After the lapse of a few days the cilia fall off, the embryos then assuming the character of planaria-like creeping larvæ.

9. Notwithstanding its abridged locomotive powers, the non-ciliated larva sooner or later gains access to the body of an intermediary bearer (the *Limnæa trunculata* or the *Limnæa druncatula*; the *Dicrocoelium lanceolatum* has for intermediate host the *Planorbis marginata*; both are common fresh-water molluscs), within whose tissues it becomes transformed into a kind of sac or *sporocyst*. In this condition the larva is capable of developing other larvæ in its interior by a process of budding. The sporocysts vary in character, and when highly organised are called *redia*; they are often also called *nurses*; the latter term being generally applicable to all forms of trematode larvæ which reproduce by internal budding.
10. The progeny of the more highly organised “nurses” (sporocysts or *redia*) are furnished with tails, in which characteristic stage of growth they constitute the well-known *cercaria* or higher trematode larvæ. In this stage they migrate from their intermediary molluscan hosts, and pass into the water to lead for a time an independent existence.
11. The pupæ or encysted cercariæ are at length passively transferred, along with its fodder or its drink, into the digestive organs of the ultimate host; and it is thought that the cysts serve the purpose of a protective covering until the larvæ have passed into the true or digestive stomach, in which organ the action of the gastric juice, by dissolving the sac, liberates the *pupæ*. From the stomach the tailless larvæ succeed in entering the common liver duct and its branches, in which situation they rapidly acquire all those internal organs which characterise the adult flukes. In this way the life cycle is completed.

From the researches of Van Beneden, and especially also of Pagenstecher, it would further appear that the degree of multiplication of the larvæ and the extent of their organisation are largely affected by varying states of the season, in association with other co-ordinating external conditions. This conclusion is important practically. For example, the highly organised germ-sacs or sporocysts (*redixæ*) are capable, under favourable climatic states, of developing not only the ordinary tailed cercariæ in their interior; but also new germ-sacs. It would seem, further, that there is no recognisable limit either to the variety or to the extent of larval fluke development. No wonder, therefore, that an accidental concurrence of favourable conditions, such as happens in particular seasons, should be followed by the disastrous outbreaks recognised as epizootics of *rot*. As we cannot regulate the character of the seasons, so neither can we prevent the occurrence of many epizootics. Helminthological science, however, does enable us to lessen the amount of disease, by affording an adequate insight into the nature of the causes concerned in its production.

The *Distoma hepaticum* and the *Dicocælia lanceolatum* are the two species of fluke found in the liver of sheep, &c. The former is distinguished from the latter by the absence of the small spines in the cuticle.

The knowledge, for instance, that the snail (*Limnæa trunculata*) harbours the cercaria of the fluke points to the necessity for the destruction of these molluscs in places subject to fluke disease, as an easy means of eradicating the infection, in the same way that the destruction of mosquitoes prevents the spread of malaria in man.

Guided by indications of the order just mentioned, Dr. Rowe, of Mount Battery, Goulburn District, Victoria, has proposed a somewhat rough-and-ready method of stamping out the rot, which disease, by the way, appears to be far more constant as an epizootic in Australia than it is with ourselves. In brief, the plan suggested was to burn the whole of the grass where rot-affected sheep have been pasturing, to destroy the diseased animals themselves, and, after a time, to restock with sound animals.

There can be little doubt that this method would prove effective for a while; but since, to insure a permanent result,

a frightful sacrifice would have to be made in the first instance, it is to be feared that the experiment could not be undertaken without severe and rather unconstitutional legal enactments. Moreover, supposing the colony were rendered entirely free from *rot*, the original exciting cause, which we are told brought the malady into the country somewhere about the year 1855, might again reintroduce the disorder. On this subject, however, the reader will do well to consult the notice of Dr. Rowe's observations, as recorded in the *Veterinarian* for February, 1873. In perusing the communication in question, it should be borne in mind that the observations proceed from the pen of an observant and extensive stock-owner.

#### BILHARZIOSIS.

This disease is caused by the presence of a parasite of the same family as fluke, or *Distoma hepaticum*. Montgomery, in India, has lately drawn attention to the prevalence of the parasite schistosomum in animals in that country. He was unable to trace any pathogenic lesions as a result of their presence in equines and bovines; but Baldrey, in the Punjab, has shown that their presence in sheep is clearly associated with a pernicious anæmia and ulcerations of the intestines. The mature worms, unlike the other trematodes, are not hermaphrodite, but have a separate male and female. They usually inhabit the blood-vessels of the mesenteric region, and their presence is liable to be overlooked, as they are very small and difficult to isolate in the vessels. Their presence may, however, be more certainly known by the demonstration of their characteristic eggs, which always show a spinous extremity at one end, rarely at the side.

The adult female lays her eggs in the mesenteric vessels, from which they become deposited in submucous intestinal or rectal tissue; here they tend to set up irritation, which may result in ulceration. They are finally expelled, and the larvæ hatch in fresh water. The eggs may sometimes be found by an examination of the fæces, or by inserting a long, blunt spoon into the rectum and gently scraping the mucous membrane. In cases where they exist, at a *post mortem* examination they can always be found by examining a

scraping of the deeper mucous lining of the intestine, usually at some ulcerated part, with a magnification of 50 to 100. Their further life-history is unknown. In man they set up a train of dangerous symptoms characterised by hæmaturia. This is due to the fact that the mature worms are generally in the rectal veins, and the eggs find their way to the submucous tissue of the bladder, from which point they are expelled. The eggs will not hatch in urine, but require admission to fresh water before doing so. This phenomenon may easily be observed under the microscope with fresh schistosomum eggs. The mature female, having expelled all her eggs, dies; and it

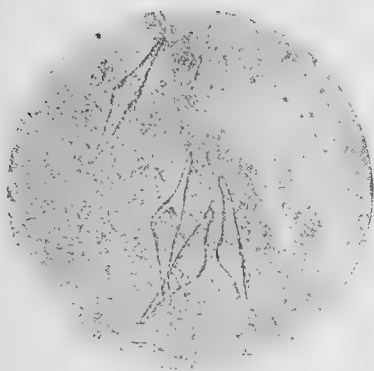


FIG. 84.—Scraping of a hæmorrhagic area, showing ova of *Schistosomum spindalis*.  $\times 50$ .

Found in mesenteric vessels of cattle.

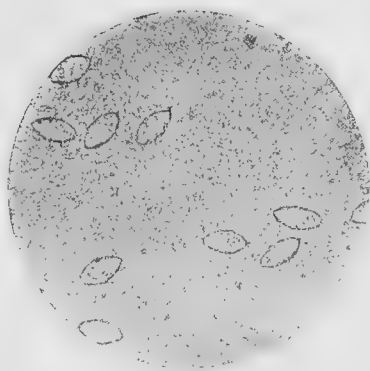


FIG. 85.—Scraping of a hæmorrhagic area, showing ova of *Schistosomum Bamfordi*.  $\times 50$ .

From mesenteric vessels of bull.

is often the case that many eggs may be found in the vessels, but no mature parasites. It is probable that the intermediate life-history of bilharzia is similar to fluke, and that infection takes place in the same way. The male is wider than the female, and the ventral sucker is very prominent; he carries enfolded in his body the female, in the so-called gynæcophoric canal. The anterior and posterior extremities of the female are free, and she is narrower than the male. It is possible that *Bilharzia crassa* is the same in animals as in man, and this constitutes a distinct danger to human beings.

The male schistosomum is 14 mm. long; the female, 20 mm. long. The eggs are 0.16 to 0.18 mm. long.

## CESTODA.

This natural order of flat-worms has acquired its name from the circumstance that most species in the adult condition resemble a tape or band, hence also the common name tape-worm. Sometimes, however, the resemblance to a tape fails altogether, as is conspicuously the case with the little tape-worm of the dog (*Tænia echinococcus*). The absence of a mouth and of an alimentary canal are very characteristic of the order, so that in the tape-worm we have an animal feeding by the process of osmosis.

If an adult tape-worm be examined we find that it consists of segments. The anterior segment is differently constructed from all the rest, and is variously designated as the *head*, *nurse*, or *scolex*. Behind the anterior segment or *head* follows a chain of segments for reproductive purposes, the *generative segments* or *proglottides*. These generative segments are produced by a process of budding from the *head*. In the same worm we find them at all stages of development, unripe, half-ripe, or completely ripe segments, the youngest nearest the head. It is quite important to observe that the *head* produces all the rest of the worm by a process of budding, so that if all the body be removed and the head left behind, the worm will be again reproduced.

The *head* or *scolex*, in addition to its power of continually budding off chains of segments, is provided with organs by means of which it fastens itself to the wall of the alimentary canal. These organs of attachment in the *Bothriocephalus* take the form of two longitudinal grooves, hence the name, while in the *Tænia* the sides of the head are provided with four sucking discs. In addition to the four suckers, some tape-worms (*Tænia armata*) have circles of hooks anterior to the suckers, each hook formed in a little pocket. These hooks have sharp projecting points, which serve as anchors for the tape-worm. Their presence or absence, their size, form, and number, are often very characteristic, and their examination is often facilitated by the use of caustic potash, in which the hard chitinous material of which they are composed is insoluble.

Following the head come the youngest segments, as yet narrow and short, and not plainly distinguishable from one another,



often spoken of as the *neck*. Proceeding backwards from the *neck*, the segments become broader and longer, and more distinct from one another. The hindmost segments have reached the full size, are capable of separating from the others, and can even have an independent existence for a time. Ultimately these detached segments wander out of the body, or are expelled with the fæces, when they die and decay; but the ripe eggs provided with their hard shells retain their vitality, and are ready, under favourable conditions, to undergo a series of metamorphoses, whose final term is the adult tape-worm.

Each segment is provided with a complete set of male and female reproductive organs, so that, if we take into account the fact that the segments can detach and lead for a time an independent existence, we may regard each *segment* or *proglottis* as a complete hermaphrodite individual, although another segment is necessary to perform the act of fecundation. In the youngest segments the sexual organs are not yet developed, but in the half-ripe segments the male and female organs are distinguishable, whereas in the completely ripe segments the uterus loaded with its eggs is the conspicuous part. The form of the uterus of the ripe segment is very characteristic, and may be readily observed by pressing the ripe segments between two slips of glass, and examining with the naked eye or a pocket lens. For example, in the *Bothriocephalus* the uterus has the form of a rosette, whereas in the *Tænia* it is usually tree-like, and by counting the number of uterine branches we are often able to determine the species. At the same time the sexual openings can be observed—(1.) On the margin, and alternately right and left, *i.e.*, on the left margin of one segment, on the right margin of the next, on the left margin of the third, and so on, *e.g.*, *Tænia cænurus*, *Tænia serrata*, *Tænia marginata*, and most *Tæniæ*; (2.) Two marginal openings for each segment, one on the right margin and one on the left (only in *Tænia cucumerina* and *Tænia expansa*); (3.) Openings not marginal, but in the middle of the ventral surface, *e.g.*, *Bothriocephalus*.

If the eggs from the uterus of a ripe segment of a *Tænia* be examined, they appear to the naked eye as coloured points, and under the microscope as round or oval bodies. The shell is

thick, and is seen to consist of little prisms cemented together. In the interior of the shell the embryo is visible as a little solid body provided with six hooks. The eggs of the *Bothriocephalus* present quite a different appearance.

These eggs are hatched when they gain access to the alimentary canal of a suitable host, which is rarely the same as the host of the adult *Tænia*. Stimulated by the warmth and acted upon by the gastric juice of the alimentary canal, the shell is more or less dissolved, and the *six-hooked embryo* or *Proscolex* becomes free. Helped by its hooks, the embryo bores its way through the wall of the alimentary tract, and is carried along with the blood, or otherwise, to some place suited for its further development; it may be the connective tissue, muscles, liver, lungs, or even the brain, as is the case with the *Cœnurus cerebralis* in the brain of sheep. Having reached a suitable resting-place, the solid embryo drops its hooks, and, acting as a foreign body, causes local exudations and new formations of granular matter and connective tissue. Thus the surrounding tissues form nutritive material and a connective tissue capsule for the solid embryo. The embryo, fed at the expense of the surrounding tissues, rapidly grows in size, loses its solid character, and becomes a hollow sac filled with fluid. In this condition the creature is spoken of as a *cystic-* or *bladder-worm*, or more commonly as a *measle* or a *hydatid*, and this cystic stage is quite comparable with the sporocyst of a trematode-worm. The cystic-worm, still enclosed in its capsule, undergoes further important changes. At one or more points in its wall a process of budding takes place, each bud growing inwards in the form of a hollow pocket, in the interior of which the suckers, or suckers and hooks, are developed. Each bud has now the form of an invaginated tape-worm head, and when evaginated is quite like that of the adult tape-worm, differing mainly from it in being hollow.

In other cases the cystic-worm undergoes more complex changes, the buds developing not into tape-worm heads, but into secondary cysts, while the buds on the secondary cysts become the tape-worm heads, as is the case in *Echinococcus* cysts.

These different kinds of cystic-worms, which are merely the larval stages of tape-worms, have received different names.

- (1.) *Cysticercus*—cyst filled with serum, and provided with only one head.

- (2.) *Cysticeroid*—cyst without serum, only one head.
- (3.) *Cœnurus*—cyst filled with serum, and provided with many heads.
- (4.) *Echinococcus*, primary cyst, gives rise to secondary cysts filled with serum, each secondary cyst producing numerous heads.
- (5.) *Acephalocyst*, without heads.

Thus the tape-worm egg, when hatched, gives rise to a six-hooked embryo, the six-hooked embryo changing into a cystic-worm, with one or more tape-worm heads, and that is the end of one part of its development.

The cystic-worm having developed its heads, can remain for a length of time without undergoing further change, cases being known in which human beings have carried an *Echinococcus* cyst for over thirty years. But if flesh, lung, liver, or brain containing cystic-worms be eaten by certain animals, then a new course of development begins. The head of the young worm comes out of the cyst, which is digested by the gastric juice, the head being protected from the action of the juice by the carbonate of lime with which it is loaded. The head is now free, attaches itself to the intestinal wall, and begins rapidly to form a chain of segments, which develop their sexual organs and their eggs, and thus we have now the cystic-worm changed into an adult *Tænia*, with its head and segments. The ripe segments containing the eggs detach and get out of the body, and now the eggs are again ready to commence anew their life cycle.

The development of the *Bothriocephalus* differs in several respects from that of the *Tænia*. The egg-shell is provided with a lid. In its interior the embryo is formed, the outer layer of cells forming the ciliated coat, and the central mass of cells the six-hooked embryo. The ciliated embryo comes out of the shell by the opercular aperture, and by means of its cilia swims about in the water. Experiments have shown that the embryos of *Bothriocephalus latus* can develop into a sexually mature worm without passing through a cystic stage. The eggs were taken and kept for six months in fresh water till the embryos were formed. A very young pup was supplied with milk containing these eggs and the ciliated embryos. On the 10th July the pup was fed with the eggs, and on the 25th August it passed a

*Bothriocephalus latus*  $42\frac{1}{2}$  cm. long—that is, about 17 inches. On the 9th September it was killed, and three *Bothriocephali* were found in its intestines.

Interesting as are the phenomena connected with the origin and development of the various tape-worms found infesting our domesticated animals, it is not necessary for the practitioner of veterinary medicine to acquaint himself with the natural history details of all the forms which are apt to come under his notice; nevertheless he will find it useful to have a general knowledge concerning some of them. Thus, he should know that no less than six different kinds of tape-worm infest the dog. Of these, he should be further aware that one (*Tænia serrata*) is acquired by swallowing the larvæ (*Cysticercus pisiformis*) which reside in the bodies of rabbits and hares; that another (*Tænia marginata*) is obtained by devouring the larvæ (*Cysticercus tenuicollis*) which reside in the viscera of the sheep and pig; that another (*Tænia cœnurus*) is obtained by ingesting the larvæ (*Cœnurus cerebralis*) which infest the brains of sheep and other animals; that another (*Tænia echinococcus*) is developed when the dog swallows the larvæ (*Echinococcus veterinorum*) found in the viscera of various animals, especially the pig; and lastly, that yet another (*Tænia cucumerina*) results from swallowing the dog louse (*Trichodectes latus*), or the dog flea (*Pulex serraticeps*), which harbours its representative cysticercus or measle.

To describe minutely all the changes through which these and other tape-worms allied to them pass, would require a separate treatise. We are concerned chiefly to present a general view of the subject; and, perhaps, in further illustrating the phenomena of tape-worm life, we cannot do better than select that particular species which, in its larval state, gives rise to the familiar disease, variously termed vertigo, gid, staggers, and sturdy.

Practical veterinarians have repeatedly asked in what manner sheep become affected with gid. For them it is not sufficient to be told that the sheep obtains the worms from the dog on the one hand, and that the dog in its turn obtains tape-worm from the sheep. There are, as we have seen, several tape-worms liable to reside in the dog, and only one of these cestodes is concerned in the matter at issue, and that is the *Tænia cœnurus*.

If the head of a yearling affected with gid be opened, one or

several hydatids will be found in the brain. These hydatids must not be confounded with the common and often much larger hydatids found in the viscera of various animals, nor with the slender-necked hydatids liable to infest the abdomen of the sheep. The brain-hydatids in question are *cœnuri*, being readily recognized by their polycephalous character; that is to say, they are furnished with numerous processes termed heads. The full-grown gid hydatid is always lodged within a sheath or cyst, and when removed, whether by operation or *post mortem*, invariably displays these heads at the surface. To the naked eye, indeed, the heads may merely exhibit the appearance of minute whitish granulations, especially if they happen to be inverted and retracted within the walls of the hydatid. The *cœnurus* otherwise presents the general appearance of an ordinary bladder-worm, containing in its interior a clear, amber-coloured, watery fluid.

On subjecting the so-called heads to microscopic examination, they will be found to display a double crown of minute hooks in front, besides four sucking discs, such as are commonly seen on the head of an ordinary tape-worm. A single large *cœnurus* may support several hundreds of these heads; each head in reality representing a young tape-worm. This relationship has been proved by experiment. If, for example, a fresh *cœnurus* be given to a dog, each of the hundred or more heads becomes converted into a tape-worm, having characters quite distinct from all the other tape-worms that are liable to infest the dog. About eleven weeks would be necessary for the tape-worm to assume its perfect or sexually mature condition. Thus, in one experiment, where only five days were allowed to elapse before the canine bearer was killed, the heads were found alive, and separated from the hydatid, but displaying no trace of any body. In a second experiment, where three weeks elapsed, the young and sexually immature tape-worms had only attained the length of one inch and a half. In a third experiment, where the interval extended to two months, the tape-worms had acquired a length of eighteen inches; but the eggs, even then, were not perfectly developed.

A period of three months being somewhat more than sufficient for the maturation of the gid tape-worm, the perfect ova will by this time be found escaping from the dog along with its

fæces. These eggs, in common with those of other tape-worms display six-hooked embryos in their interior. Wherever the infested dog wanders and passes excrement *per anum*, there will it be privileged to distribute the ova. The eggs, if left to themselves, would do no harm; but by various agencies they are further distributed over the pastures where yearlings and sheep are grazing. Millions of tape-worm germs are thus annually scattered far and wide. In due course the ova are swallowed by grazing animals. When the ova have arrived within the true digestive stomach, the gastric juice dissolves the shells, and the minute six-hooked embryos forthwith make their escape. They speedily set about migrating on their own account; and having, by means of the hooks in question, bored their way into the blood-vessels, they are carried to and fro in the current of the circulation. By virtue of some selective capacity they seem to know when they have arrived within the vessels of the brain, in which organ, after escaping the vessels, they bore their way to the final resting-place. Here, by a process of transformation, they part with their hooks, and gradually acquire the bladder-worm state, in which condition they vary in size from a pin's head to that of a large walnut. To attain the perfect polycephalous state, they require a period of about ten weeks, and thus the whole cycle of development is accomplished within something like five months.

It must be allowed that the process of development above recorded is one of the most astonishing of all the biological phenomena with which the naturalist is acquainted. Not merely are the necessary changes of host remarkable, but the characteristics marking each phase in the life-history of the entozoon itself are still more noteworthy. At one time of its career the creature is a mere bladder-worm, at another an elongated tape-worm, at another a minute six-hooked embryo, and finally, once more, a bladder-worm. But this is not all; inasmuch as its development, in one very important particular, differs from the process undergone by the ordinary beef and pork tape-worms (*Tænia mediocanellata* and *Tænia solium*). Thus, whilst the egg of the common tape-worm is only capable of developing onward into a single sexually mature tape-worm, the solitary egg of the *Tænia cœnurus*, as we have seen, becomes transformed into a multitude of tape-worms. In this respect,

it is true, our parasite is, in some sense, eclipsed by another tape-worm that is resident in the dog (*Tænia echinococcus*); but, with this exception, we know of nothing comparable to it within the limits of cestode reproduction.

Amongst the other tape-worms of general interest to the professional man are those which are derived from the consumption of pork and beef. The measles of pork (*Cysticercus cellulosus*) are transformed into the *Tænia solium*, whilst those of beef (*Cysticercus bovis*) are transformed into the *Tænia mediocanellata*. These tape-worms, as such, are only known to infest the human body. The tape-worms of cattle and sheep, as well as those of the horse, probably all belong to that group of tape-worms whose larvæ are normally resident in the bodies of insects and other non-vertebrated animals. This explains why their separate developmental histories have not hitherto been fully made out by helminthologists.

Those who desire further details on this head should consult the standard treatises of Leuckart and Küchenmeister, Neumann's "Parasites," or Cobbold's manual of the parasites of our domesticated animals, in which the medical and sanitary importance of the beef tape-worm is dwelt upon at considerable length. Lastly, we desire to call particular attention to the fact that cysticerci or measles have been found in mutton. These small cystic-worms are undoubtedly the representatives of a distinct species of tape-worm that probably resides in the human bearer. Some account of the parasite (*Cysticercus ovis*) is given in the supplement to Cobbold's larger treatise on *Entozoa* (p. 30), the selfsame entozoon being much more recently and fully described by Dr. Maddox in the *Monthly Microscopical Journal* for June 1873. We now know, therefore, that mutton and beef, as well as pork, may become measled; these three kinds of measles being perfectly distinct from each other, and all severally derivable from different species of tape-worm. It is now known that these ovine cysts were undeveloped *Cysticercus tenuicollis*, and that the *Tænia tenella* of man was a small-sized *Tænia solium* or *Tænia saginata*.

## ACANTHOCEPHALA,

As already stated, this order of helminths is represented by a parasite which occasionally takes up its residence in the intestines of the pig. It is known as the large thorn-headed worm, or *Echinorhynchus gigas*. The male commonly measures three or four inches in length; whilst the female often exceeds fifteen inches, examples having been recorded beyond two feet. The head is furnished with an armed proboscis, by means of which the worm anchors itself securely within the mucous membrane of the small intestines. This parasite is tolerably abundant in France and Germany, but very little is known of it in England. Professor Verrill, writing for the "Report of the Connecticut Board of Agriculture," speaks of this parasite as "the commonest and most injurious intestinal worm found in swine. These parasites," he adds, "not unfrequently perforate the walls of the intestine, and stray into other parts of the viscera, producing serious disease. Sometimes the intestine of a hog is found perforated by so many of these holes that it cannot be used in the manufacture of sausages. In severe cases the hogs are weak in the loins, and have the membranes in the corners of the eyes swollen, watery, and lighter coloured than usual. The excrement is hardened and highly coloured, and the animal often keeps up a continual squealing and grunting, especially in the morning. Such hogs are generally cross and morose, biting and snarling at their companions, but usually too weak to defend themselves if attacked in return, and easily thrown down. Finally, the weakness increases until the poor creatures are unable to walk about or to stand."

Although the development of the *Echinorhynchus* of the hog has never been fully traced, there can be little doubt that its mode of development is precisely similar to that known to occur in other members of the genus. Dr. Guido Wagener has furnished us with some admirable illustrations of the eggs and embryos of various species (Sieb. and Köll., *Zeitsch.*, vol. ix.), but it remained for Leuckart to explain that these creatures during growth exhibit the characteristic phenomena of alternate generation (*Gött. Nachrichten*, 1862). His experimental investigations were chiefly made with *E. proteus* and *E. filicollis*. Professor Leuckart caused some fresh water-crustaceans (*Gammar*) to



swallow the eggs of these small thorn-headed worms, and he had the satisfaction of observing that in a few days the embryos quitted their egg-shells and passed into the bodies of the unsuspecting intermediary bearers. After a series of further changes (which Leuckart regarded as comparable to those of a true alternate generation, and not simply metamorphic), the young parasites rapidly increase in size; the original skin of the embryo being cast off "as soon as the *Echinorhynchus* occupies the whole interior of the embryo." The young parasites acquire sexual organs whilst still lodged within the intermediary bearers, so that, within about a week after they are transferred to the intestinal canal of their proper and ultimate piscine host, their development into the adult state is completed. What ordinarily takes place in the case of these echinorhynchi of the fish must more or less appertain to the echinorhynchi of the hog. Swine, as we all know, are not very particular as to what they eat or drink, consequently they have abundant opportunities of swallowing insects, gammari, entomostracous crustaceans, or other minute creatures which are destined to harbour the larvæ of acanthocephalous parasites.

According to Schneider the eggs of the *Echinorhynchus gigas* are discharged in the fæces of the pig, which harbours the sexually mature adult. The eggs are devoured by maggots, and, reaching the stomach, are hatched. The embryos, which are provided with spines, bore their way into the body cavity of the maggot where they develop a young *Echinorhynchus* in their interior. The maggots are in turn devoured by the pig, in which the *Echinorhynchus* again reaches sexual maturity and produces ova.

#### DIPTERA.

As remarked at the commencement of this chapter, some of the flies are apt to prove troublesome as internal parasites. With those dipterous or two-winged insects which, as *external parasites*, occasion suffering to animals, we have here little or nothing to do; but since some of these forms of insect life play the double part of attacking their victims from within as well as from without, it is desirable to speak of such of them as fairly, in one phase of their life, come under the general class of internal parasites.

In this relation there is a particular group of insects that distinguishes itself above all the others. This is the so-called bot-producing family, comprising various forms of gadflies (*Æstridæ*).

As enemies of the horse, ox, and sheep, the gadflies have acquired notoriety from the earliest times. Thus they originally obtained their family title from the ancient Greeks, who called the gadfly of cattle the *Oistros* (Οἶστρος, from Οἶω, I impel; Latin, *Æstrus*).

When any person became unduly excited they said he had a fit of the *Oistros*. One can readily see the force of this expression after noticing how outrageously excited and furious a herd of cattle becomes when attacked by gadflies. As, however, it is not with the gadflies, viewed in the light of external enemies or parasites, that we have here to deal, we may dismiss this part of the subject by observing that, in the case of cattle, the gadflies have the ingenuity to select as their victims young beasts from two to three years old. The hide of an older beast is more difficult to pierce.

Since different species of gadfly attack different animals, and several kinds of fly, in the larval state, infest one and the same animal, it is desirable to speak of the forms belonging to our various domestic animals separately. In the first place we will consider those of the horse.

The common gadfly of the horse (*Æstrus equi*) attacks the animal whilst grazing late in the summer; its object being not to derive sustenance, but to deposit its eggs on the coat; and this it accomplishes by means of a glutinous material causing the ova to adhere to the hairs. The parts of the animal selected are chiefly those of the shoulder, base of the neck, and inner part of the fore legs, especially about the knees; for in these situations the horse will have no difficulty in reaching the ova with its tongue. When from any cause the animal licks those parts of the coat where the eggs have been placed, the moisture of the tongue, aided by warmth, hatches the ova, and in something less than three weeks from the time of the deposition of the eggs, the larvæ thus make their escape. As maggots, they are next transferred to the mouth, and ultimately to the stomach of the equine bearer along with food and drink. Of course a great many larvæ perish during this passive mode of immigra-

tion; some being dropped from the mouth, and others being crushed in the fodder during mastication. It has been calculated that out of the many hundreds of eggs deposited on a single horse, scarcely one out of fifty of their contained larvæ arrive within the stomach. Notwithstanding this waste, we are all of us familiar with the circumstance that the interior of an animal's stomach may become completely covered with the larvæ in the condition of "bots." Whether few or many, they are retained in this singular abode chiefly by means of two large cephalic hooks, which are inserted into the mucous membrane.

As soon as the bots have attained their perfect growth, as such, they voluntarily loosen their hold, and allow themselves to be carried along the alimentary canal until at length they make their escape with the fæces. It is said that during their passage through the intestinal canal they not unfrequently re-attach themselves to the mucous membrane, thereby occasioning severe intestinal irritation. When thus lodged in the neighbourhood of the anus they seriously inconvenience the animal. In all cases, however, they sooner or later fall to the ground. When once transferred to the soil they bury themselves beneath the surface in order to undergo the change whereby they are transformed from the bot state into the pupa condition. At length, having remained in the soil for a period of six or seven weeks, they finally emerge from their pupal envelope or cocoon in the active life-phase of the imago or perfect dipterous insect. It thus appears that these creatures in the form of bots ordinarily pass about eight months of their lifetime in the digestive organs of the horse.

That bots are capable of giving rise to severe disease in the horse there cannot be any reasonable doubt, but it is not often that the disorder is correctly diagnosed, since it is only by the passage of the larvæ, or by their adherence to the verge of the anus, that the practitioner can be made aware of their presence. We are not called upon to dwell on this fact of the subject in the present chapter, but may remark in passing that Mr. J. S. Woods, V.S., has published in the *Veterinarian* a case of tetanus in a mare associated with the larvæ of *Æstrus equi*, and Mr. J. T. Brewer, V.S., has also given a case in the same journal, where the duodenum of a horse was perforated by bots.

Several other species of *Estrus* victimize the horse ; one of the most formidable of these being the *E. hæmorrhoidalis*. This fly is especially annoying in the initiatory stages of the attack, because, unlike the common species, it selects the lips and nostrils as the principal locality for the lodgment of the ova. According to Bracey Clark the mere sight of the insect produces extreme agitation, the horses wildly galloping to and fro in their usually vain endeavours to evade these winged tormentors.

The common bot-fly of the ox (*Estrus bovis*) passes through transformations similar to those undergone by the gadflies of the horse. It differs, however, in one important particular ; for, in place of acquiring its larval condition as a bot within the stomach, it takes up its residence for that purpose beneath the animal's hide. In this situation its presence gives rise to the formation of small tumours, termed *warbles*. The facts, in short, are as follows :—Selecting, as before remarked, young beasts in good condition, the fly lights on the back on either side of the spine. The animal darts away in alarm, often bellowing furiously and frightening its companions. The whole herd forthwith rush about in a frantic manner ; and it is said that the mere buzzing of the insects is sufficient to render yoked animals quite unmanageable. In a short time the insect succeeds in perforating the skin by means of an ovipositor, one egg being deposited in each opening. After a time the egg is hatched, and the young during growth produces sufficient inflammation to lead to the formation of the well-known *warbles*. Within the tumour the bot is placed with its head downwards, its tail being applied to a small external opening in the warble, in order that it may receive sufficient air for the purposes of respiration. When the bots are mature they make their escape, and fall to the ground, burying themselves in the turf, or hiding underneath stones. During the process of metamorphosis the skin of the bot becomes transformed into a cocoon, and in course of time the pupa or chrysalis stage is completed. In this state it remains as a grub for a month or six weeks, at the expiration of which period the lid of the cocoon comes off, and the perfect insect or imago is set free.

The common bot-fly of the sheep (*Estrus ovis*) neither chooses the stomach nor the back of its bearer as a place of residence during its acquisition of the larval condition, termed the bot.

This insect may be regarded as a worse tormentor than either of its common congeners above mentioned. It attacks the nostrils of the sheep, and the distress thus occasioned is so great that the poor animals, in order to avoid the flies, will often bury their nostrils in the dusty hollows of cart-ruts, further protecting their heads with the fore feet. The members of any flock thus attacked will also collect together in groups, and jostle against one another with their heads downwards, so as to avoid the flies as much as possible. When struck by the fly they stamp the ground violently, and exhibit other signs of distress, sometimes amounting to agony. According to Mr. Riley, as quoted by Verrill, the young larva is itself deposited at the margin of the sheep's nostrils, having quitted the egg whilst yet within the oviduct of the parent insect. Be this as it may, the young larvæ, having once gained access to the nasal passages, have no difficulty in retaining their hold, at the same time that they cause fresh distress to the unhappy bearer. Within the frontal sinuses they firmly anchor themselves by means of a pair of cephalic hooks, and in this situation they remain until they have perfected this stage of their larval development. Considering the situation of these creatures, there need be no astonishment at the fact that their presence sometimes gives rise to terrible sufferings on the part of the sheep; the afflicted animals occasionally perishing under the inflammatory action thus set up. Stock-owners and farmers term this disease grub in the head, and it is often asserted by them that the grubs gain access to the substance of the brain itself. They are perfectly sure they have seen maggots in the brain, and no arguments of the veterinarian, derived from a study of the osteology of the sheep's head, will serve to convince them that they are in error. Without dwelling upon this point, we have further to observe that the perfected bots usually pass from the nostrils to the ground by the same way that they entered, and thenceforward, having penetrated the soil, they accomplish their subsequent metamorphosis in a manner very similar to that of their congeners. The pupal state is acquired in about two days, but they remain concealed in the soil for a period of six or eight weeks. At the expiration of this period the lid of the cocoon is raised, and the insect prisoner makes its escape in the usual manner.

In perusing the above remarks, it will be noticed that, if Mr. Riley's statements are to be accepted as correct, the gadfly of the sheep reproduces viviparously. This is a point of considerable interest, since, so far as we are aware, all the other gadflies bring forth their young in the egg condition. According to Verrill, who quotes from the *First Annual Report on the Noxious Insects of Missouri* (given in the Connecticut publication already cited) for 1868, Mr. Riley states that Mr. Cockrill had removed upwards of three hundred living larvæ from the body of a single gadfly. Soon after the flies have effected their escape from the cocoon, they set about operations for the continuance of the species; and as they are neither furnished with a mouth nor other means of taking in nourishment, it is obvious that the pleasures they enjoy during the winged state must be exceedingly short-lived.

Of late years, since the knowledge of protozoan diseases has improved, the importance of flies of the order Diptera has greatly increased from the point of view of economy. It is now known definitely that the varieties of disease due to trypanosomata are spread by various dipterous insects, the more important being that family known as *Glossina* and the family *Tabanadæ*.

The *Glossina*, or tsetse-flies, have nine species. They are greyish or yellowish-brown, dull-coloured flies 5-7.75 mm. long. During life and while at rest their wings are characteristically crossed over the back, exactly like the blades of a pair of scissors. Metamorphosis is incomplete or pupiparous—i.e., larva, pupa, adult. There are no eggs. The fly is exclusively confined to Africa, except one species, the *Glossina tachinoides*, in Arabia. The *Glossina palpalis*, *fusca*, and other species convey sleeping sickness. *Glossina morsitans* is the agent conveying nagana.

*Stomoxys calcitrans*, or stable-fly, is common in England, but it is not known to produce disease. It has four stages in its life cycle—egg, larva, pupa, and imago, or adult. Eggs are laid in manure heaps, etc., usually in large quantities together, so that careful search will render their destruction easy; moisture is absolutely essential for their existence.

*Tabanadæ* (horse-flies, clegs, breeze-flies, etc.). Usually a large and ferocious fly measuring from a half to one inch

long. Eggs are laid on leaves of plants in clusters, and these develop into larvæ and pupæ. A species of this fly is concerned in the transmission of surra in India.

*Hippoboscidae* resemble *Glossina* in their metamorphosis. They are known as forest-flies, and are said to be the cause of the spread of disease.

It has been known or suspected for some time that the common house-fly (*Musca domestica*), or the blow-fly (*Calliphora vomitaria*), can be the mechanical means of conveying disease from animal to animal, by the mere fact of carrying disease-producing germs on their feet and depositing them on a sore or on some food material.

*Ticks* have of late become of very great interest to the veterinarian, since it is known that they are active agents in the spread of piroplasmosis and spirillum fever. Formerly they were considered as parasites, simply causing annoyance and harm to the skins, wool, food, &c., but now there are graver reasons for their systematic study.

The *Ixodoidæ* or tick family belong to the *Arachnoidæ*, a family of the *Arthropodæ*, and are closely allied to the spiders and scorpions. They are of the sub-family mites known as *Ixodidæ*.

The *Ixodidæ* are divided into the *Argasidæ*, having no scutum, and the *Ixodidæ*, having a scutum. Of the *Argasidæ* there are only two families, the *Argas* and the *Ornithodoros*, the former having the rostrum entirely invisible from above, the latter having at least the tips of the palpi visible.

The *Ixodidæ* are divided into the *Rhipicephalinæ*, having the rostrums shorter than broad, and the *Ixodidæ*, having the rostrum longer than broad. The former are, again, divided into *Boophilus*, *Rhipicephalus*, *Hæmaphysalis*, and *Dermacentor*; the latter into *Ixodes*, *Hyalomma*, *Amblyomma*, and *Aponoma*.

The differentiation into species from these groups is unnecessary here, and we need only mention the chief species concerned in spreading disease.

*Rhipicephalus appendiculatus* conveys East Coast fever.

*R. annulatus*, Texas fever.

*R. Australis*, red-water, in Australia. The infective parasite is transmitted through the mature female to the larvæ, which are alone capable of transmitting the disease.

Bilious fever of the horse by probably *Hyalomma Egyptium*.

Bilious fever of the dog by *H. Leachii*.

Heart-water in cattle by *Amblyomma hebraeum*.

Hæmoglobinuria in Europe by *Ixodes reduvius*.

"Trembling," or Louping-ill in sheep, *Ixodes reduvius*.

Spirillum fever by *Ornithodoros moubata*.

In the dog only the adult tick conveys the disease, the larvæ and nymph being innocuous.

In East Coast fever the ticks contract the infection as larvæ or nymphs, and convey it to cattle when they are nymphs or adults.

All ticks are very resistant, especially the Bont tick in South Africa, which also conveys East Coast fever, and for this reason special methods of dipping and other precautionary measures are rigorously enforced.

#### TRACHEARIA.

As with the dipterous insects, so with the members of this large order or division of arachnidans. Whilst many species are externally parasitic, only a very small number can in any true sense be called entozoa. One species, however, of the present group is not only internally parasitic in the larval state, but also in the full-grown or sexually mature condition; consequently it has even more right to be regarded as an entozoon than any of the gadflies. The parasite in question is the *Pentastoma tænioides*. In the adult state, this worm occupies the nasal and frontal sinuses of the dog, sheep, and horse; and in one of its larval stages it is found either encysted or free in the viscera of the various animals, especially ruminants, as well as in man himself. The larva, which is commonly described as the *Pentastoma denticulatum*, usually measures about one-fifth of an inch in length; but the adult males are three-quarters of an inch long, whilst the females occasionally measure as much as four inches from head to tail.

The history of the development of these curious parasites is somewhat remarkable and of great practical interest. The female discharges her eggs whilst within the nose of the dog. From the nasal passages the eggs are constantly discharged by sneezing and otherwise. They are thus scattered by the canine host in



all directions, and by the drying of the slime they adhere very readily to vegetable and other matter. Afterwards, on being transferred to the stomach of ruminating and other animals, their embryonic contents are set free by the dissolution of the shells. The embryos, which are furnished with two pairs of claws, then bore their way into the liver and other viscera. They next become encysted and change their skins. After a time, the larvæ are set free, but their wanderings are ordinarily of no avail, unless portions of the infested animal are brought in contact with the nose of the ultimate bearer. In the case of the dog, this commonly happens when the animal is engaged in devouring portions of fresh viscera carelessly flung to it. The larvæ are thus brought in contact with the dog's nose, and then, by means of the hooks and spines with which they are armed, the young pentastomes readily adhere to the nose, and in a short while crawl up the nasal passages, where they rapidly acquire sexual maturity. It is not so clear how they make their way into the nasal cavities of the sheep and horse; but in all probability some larvæ escape from the bodies of their intermediary bearers into open pastures, and thence into the nasal organs whilst these animals are grazing. Dogs that frequent knackeries and slaughter-houses are particularly liable to become infested by the adult parasite. Such larvæ as do not succeed in escaping their cysts perish by calcareous degeneration.

To the veterinarian these facts of development are chiefly important as explaining how dogs contract the parasite; and although instances of suffering from these entozoa may not be common, there is every reason to believe that examples of the kind are not unfrequently overlooked. It is certain that dogs sometimes suffer severely from worms in the nose, the parasites giving rise to nasal catarrh, accompanied by foetid discharges. There is one remarkable case on record, in which death was occasioned by pentastomes. This is the instance already mentioned as having been made public by Professor Dick in the pages of the *Veterinarian* for 1840. The communication is most interesting and instructive. Three of these parasites having wandered into the fauces and trachea of the dog, its death resulted, partly from spasm of the laryngeal muscles, and partly from inflammation of the left lung, accompanied by excessive bronchial secretion. In short, the animal was suffocated.

## ASCARIDES AND LUMBRICI.

The *Ascaris megalcephala* of the horse and *Ascaris suilla* of the hog are considered by some helminthologists to be identical with the *Ascaris lumbricoides* of man. Dr. Cobbold and others take exception to this conclusion, and consider that they are distinct species.

These worms, best known amongst veterinarians as the *lumbrici*, resemble the common earth-worm in size and shape; the males are shorter than the females, which sometimes measure from twelve to sixteen inches in length. They are found in the small intestines; sometimes, but rarely, in the stomach. If few in number, they occasion no inconvenience to the bearer; but if numerous, and particularly if they infest the stomach, they, like bots, may cause colic, indigestion, unthriftiness, and emaciation. After the death of the bearer, several of them are generally found matted together, and coiled up in the form of a ball, leading one to conclude that they have thus caused an obstruction during life. Numerous observations in the dissecting room enable me to state that they assume this form after the death of the host; that they congregate together, interlace one with the other, very shortly after the animal which they infest has died.

In the dog, round-worms—*Ascaris marginata*—especially if they enter the stomach, cause convulsive fits, vomiting, and sometimes death.

*Oxyuris curvula*, or better known to veterinarians as *ascarides*, are small white worms—needle or whip worms—which commonly infest the intestine, and very often the colon (and the mucous membranes of these viscera), being very abundant in the flexures of that intestine; when mature they descend to the rectum. They escape by the anus, and cause irritation, manifested by the horse rubbing its tail against any hard substance, such as the walls of its stable, or whisking it about in an irritable manner. If the anus and perineum be examined, small masses of a yellowish-white-looking substance will be found adhering to the skin; these are the eggs of the worms discharged from the body.

*Treatment.*—It is a difficult matter to destroy these parasites. Sometimes an aloetic cathartic will cause the expulsion of numerous *lumbrici* from the horse, but the remedy is uncertain. Oil

of turpentine stands in high repute, and doubtless is more anthelmintic than any other in the horse, but it cannot always be depended upon. Aconite, in some instances, will cause the expulsion of the parasites, as is seen when it is employed in the treatment of inflammatory diseases. When associated with emaciation and debility, the salts of iron prove of much service; they not only have the effect of destroying the worms, but, by causing an improvement in the general health of the animal which they infest, render it an unfit habitat for the parasites, for it is a well-known fact that when the condition of the animal's body is weakly, it is more liable to be infested by parasites of various kinds.

In the dog, emetics will sometimes cause the expulsion of the *Ascaris marginata* when in the stomach; if these fail, santonine, in from three to five or eight grain doses, according to the size of the animal, or the ethereal extract of the male shield fern, is to be administered every second or third day, taking care to watch their action upon the patient, as santonine will sometimes cause straining and other signs of irritation.

The *oxyuris* may sometimes be destroyed in great numbers by enemas, consisting of decoctions of quassia, gentian, or even wormwood. These enemata are to be frequently repeated, and their action may be aided by purgatives. The introduction of a small piece of mercurial ointment into the rectum is a very common practice in some parts of the country, and it seems to answer very well, not only preventing the migration of the parasites, but actually destroying them.

There are many other remedies recommended by helminthologists, many of which are very serviceable, whilst others are only calculated to do harm. I need only refer to the insane practice of administering ground glass and other mechanical irritants, in order to warn the reader against what is absurd.

Other nematode worms infesting the lower animals, and found in the blood-vessels and eyes, are briefly referred to in my work on Veterinary Surgery, to which the reader is referred.

## CHAPTER LXXXIV.

### PARASITIC DISEASES—*continued*.

#### DISEASES CAUSED BY NEMATODA OR ROUND-WORMS. . . .

##### PARASITIC DISEASE OF THE LUNGS.

IN the calf and lamb, bronchial irritation, arising from the presence of nematode parasites, termed strongyles, is of frequent occurrence, and is variously termed *The Husk*, *Hoose*, *Phthisis pulmonalis verminalis*, and *Parasitic bronchitis*.

In the lamb, the parasite, termed *Strongylus filaria*, is from one to two and a half inches long; the female is white, larger than the male, which is of a yellowish-white colour, and its body is of uniform size, but tapered at both ends. The head is short, stumpy, rather angular, but not tuberculated as in other strongyles. Extending from the mouth is a short œsophagus, entering the stomach, from which a straight intestine is continued nearly to the extreme end of the tail. The tail of the female is pointed; the oviducts, filled with eggs and live young, extend into the vulva, which is situated close to the anus.

The anatomical situation of the parasites in the lungs of lambs and sheep is not always the same. In lambs they are found not only in the bronchial tubes, but also in the lung-substance, whilst in sheep they are generally encysted in the parenchyma of the lungs, giving them the appearance of being filled with small tubercular deposits; indeed, the disease was for a long time looked upon as a true tubercular affection.

The lungs of sheep which have been the hosts of these parasites are thickly covered with numerous small nodules, varying in size from a pin-head to a hemp-seed, or even larger, and resembling small vesicles or blisters. Some are filled with a

clear fluid; others contain a soft material, consisting of granular matter; whilst others are hard and gritty—and all contain minute worms coiled upon themselves.

The likeness to tuberculous lesions is enhanced in all these parasitic affections of the lungs. When examined microscopically, it will be seen that the nodule has numerous giant cells, in many of which are coiled up the small embryonic forms of the worm, or the vacuole-like space from which it has escaped. Their residence within a giant cell does not in any way interfere with their growth and progress.

The varying degrees of hardness and consistence of the nodules mark their age; the vesicle seems to be their earliest development; the soft, solid condition a more mature condition; and the gritty state shows that the wall has undergone calcification, and that the worm has been lodged in the lung tissue for a considerable period.

The presence of these parasites in the lung does not always cause irritation or inconvenience to the host. The lungs of sheep killed in the prime condition are found loaded with them. Occasionally, however, they induce debility, anæmia, and cause death, more particularly in lambing ewes, at or about the period of parturition. Many ewes died from this cause during the lambing season 1874.

In lambs, however, the parasites find their way through the softer lung structure into the bronchial tubes, and there give rise to irritation, and to the symptoms of the "lamb disease."

It is supposed that the parasite is developed in the lamb only, and that those found encysted in the lungs of sheep have been long imprisoned as it were by a boundary line of plastic inflammation, which finally becomes calcified, and offers an impassable barrier to the movement of the strongyle.

Whilst admitting the greater frequency of the affection in lambs than in sheep of a more mature age, I cannot subscribe to the above conclusion, as the results of examinations of the lungs of four or even five year old ewes have shown conclusively that many cysts are not in a state of calcification—a condition that they certainly would be in, if the parasitic invasion had occurred when they were lambs. The manner by which parasites gain access to the lungs has been a matter of controversy. Dr. Edward Crisp, in an essay on this disease, for which

a prize of £30 was awarded by the Bath and West of England Agricultural Society, accounts for their presence in the lungs by direct passage into the trachea from the mouth, where they have been forced from the stomach during the act of rumination. Mr. Dickinson, M.R.C.V.S., Boston, Lincolnshire, Professor Armatage, as well as Professor Gamgee, oppose this view. Mr. Dickinson says—"The idea entertained of a direct passage of these to the lungs appears to me paradoxical and in nowise probable. Their migration, I am inclined to believe, is a work of time; and hence, as I have frequently observed, the parasites abound in the alimentary canal in large numbers, give rise to aggravated symptoms which terminate fatally when no worms or their eggs are to be detected by the naked eye, at least in the lungs or bronchi." Professor Armatage informs me he has also observed this in many instances. Mr. Armatage says—"The presence of *Strongylus filaria* in the lungs of lambs and sheep, I think, cannot be explained by any theory which describes—no matter with what minute exactness and elaborate detail—the unnatural and, I might add, almost impossible mode of their passing direct through the windpipe to these organs. We all know, as possessing some slight knowledge of physiology, how a hair, a breadcrumb, drop of water, &c., will irritate the glottis or entrance to the windpipe, and give rise to the most painful and convulsive coughing. These are, however, objects of an inanimate character, and irritate by mere presence. How much greater, then, would be the effects of a live worm or worms insinuating themselves on the delicate structures, and especially when the natural barbs or hooklets, as described by Professor Simonds, are put in operation. The supposition, I think, suffers much under the great probability that violent coughing taking place on the entrance of a worm or worms would entirely expel them. If they are expectorated in large numbers from the recesses of the bronchial tubes, they will most assuredly be compelled to evacuate much more rapidly at their entrance to the windpipe." The same gentleman further says—"We must not overlook the important fact that young lambs are principally affected. In them the tissues are more easily pierced, and their passage from the lung tissue to the bronchial tube readily effected. In older animals they remain enveloped within a matrix of cretified substance and metamorphosed lung tissue, in order, as it were, to

guard against their effects. If their passage to the lungs were always more direct, the opposite would be the case, and our old animals would die as rapidly as the young ones." Professor Gamgee says—"The migration from the mouth or alimentary canal to the lungs certainly requires a more complete explanation than has hitherto been given."

There is no doubt in my mind but that the ova and young parasites taken up with the food, in the first place gain access from the alimentary canal into the circulation, and are conveyed into the lung-substance, where they are deposited, the parasites when mature piercing the tissues and entering the bronchial tubes, and there cause the irritation symptomatic of the disease, whilst those remaining encysted in the lung cause little or no inconvenience. It is very true that in many instances the parasites are found fully matured in the digestive canal, and doubtless the conclusions of Dr. Crisp are due to this fact. We can, however, easily understand that the heat and moisture of the stomach are quite sufficient to cause the ova to hatch and some of the embryos to mature in the intestinal canal, whilst other embryos pierce the intestinal wall and are taken into the circulation, and mature only when they have been deposited in their proper habitat; and a few may become fully developed in the blood. I have on two occasions seen the parasite in the cavities of the heart and in the blood-vessels; and I think this fact conclusively points out the correctness of this view of their migration.

The tenacity of life in the young strongyle is very great. Ercolani found that they showed signs of life on being moistened after drying for thirty days, and at other times after having been immersed in spirits of wine at 30°, or in a solution of alum and corrosive sublimate.—(GAMGEE.)

The number of embryo worms in the lungs of one sheep is very great; if to these we add those hatched in the digestive canal, we can easily understand, when these are severally discharged from the infected animal, how a pasture may become infected with parasites and ova to such an extent as to infect a whole flock.

Dr. Crisp says the disease is due to over-stocking, and especially to the feeding of lambs off a second crop of clover after the first crop has been consumed by sheep. The mere feeding of lambs with the second crop of clover, after the first has been con-

sumed by sheep, would not of itself be sufficient to cause an outbreak of the lamb disease; but when we consider that a very large number of sheep are infested with the parasites, from which they seem to suffer no harm, we can easily understand how, by the expulsion of some of these from the affected, a pasture may become fouled by the parasites and their ova.

It is more than probable that the *Strongylus filaria* is taken in during ingestion, and from the stomach or intestines finds its way to the lungs as already explained.

We know, however, that the disease prevails particularly in low, damp situations; on lands subjected to be flooded by the overflowing of rivers, or after heavy rains occurring during early autumn or the latter part of summer.

*Symptoms.*—These are subject to some modification, depending upon the seat of the parasites. In the majority of cases, they are found in the lungs, in others in the lungs and digestive organs, whilst in others again they are found in the stomach and bowels only—the lungs being entirely free from them. When in the intestinal canal the symptoms are those of dysentery, with foetid stools; there is much tenesmus or straining, and occasionally clots of foetid blood are discharged *per rectum*. The lodgment of the parasites in the pulmonary tissue and bronchial tubes cause, in the lamb, irritation and inflammation, indicated by cough, rubbing the nose on the ground, and accelerated respiratory movements, whilst in the more mature animal—the sheep—the presence of the parasite may induce no diagnostic signs, even in those which succumb, and it is only after death that the cause of the emaciation and anæmia, which may have been present during life, is discovered. In many cases the abdominal symptoms are accompanied by depraved appetite, intense thirst, and even colicky pains.

When the parasites are in both the lungs and bowels, there will be a combination of pulmonary and enteric irritation, and very rapid sinking. If the expectoration and alvine discharges be carefully examined some of the parasites are sometimes discoverable.

*Treatment.*—This naturally divides itself into preventive and curative.

To prevent the disease, lambs require to be fed on fresh pastures; second and third year crops are to be avoided, specially if they have been previously grazed by sheep. If the



seasons are damp, the flocks are to be pastured on the hill sides, or on dry pastures; and if the grass be scarce, it is to be supplemented by artificial food.

To cure the disease, or rather to destroy its cause—the parasites—inhalations of chlorine gas have been recommended. In using this agent great care must be taken that it be sufficiently diluted with air, so as not to destroy the patient as well as the parasite, for I have heard of individuals who have applied it sufficiently strong to destroy all their patients. It is therefore better to compel the animals to inhale it from the chloride of lime, to which sulphuric acid has been added, than to manufacture it in the ordinary way, namely, by the admixture of common salt, peroxide of manganese, and sulphuric acid. Should the animals be thought too weakly to stand the chlorine gas, sulphurous acid may be substituted, and this is so cheap and so much safer than the chlorine that I much prefer it. It is made by burning sulphur, which, combining with oxygen during the combustion, gives off fumes of sulphurous acid. Of course it will be understood that when animals are made to inhale either of these gases, they are to be confined in some building. When the parasites are in the intestines, several doses of turpentine are to be administered. The debility from which the animals suffer is best combated by stimulating food, as the cakes, to which the sulphate of iron—from ten to twenty grains for each lamb—has been added. Rock salt should also be allowed the animals to lick, or a small quantity of common salt added to the food.

*Hoose in Calves.*—This disease very closely resembles that in lambs; and is caused by a parasite termed the *Strongylus micrurus* (Mehlis), which gains access to the pulmonary tissue and bronchial tubes through the circulation, the ova being absorbed from the digestive canal. This parasite is very tenacious of life, and will be seen to be quite lively in the pulmonary organs several days after the death of its host. It is one of the armed strongyli, has a filiform body, and a mouth with three papillæ.

This disease prevails in low-lying districts, on land near rivers, more especially after heavy floods, and is mostly seen in the months of August, September, and even October, in calves under one year old, and very rarely in those rising two years old.<sup>1</sup>

<sup>1</sup> I found this parasite in the lungs of several full-grown American oxen during the spring of 1879.

Sometimes the worms are very numerous in the trachea and bronchial tubes, and not unfrequently one finds them congregated together, after the animal's death, in a perfect ball, effectually obstructing the windpipe.

I have watched the movements of the embryos of this parasite under the microscope, and have seen them burst through the walls in which they have been enclosed, and make their escape.

This parasite has also been found in the air passages of the horse and ass. A similar affection has been observed in the pig, the parasite being called *Strongylus suis*. In poultry the disease termed the "gapes" is caused by a parasite called *Syngamus trachealis*.

*The Symptoms in the Calf.*—The seat of the irritation is indicated by a bronchial cough, "husk or hoose," loss of flesh, a varying degree of constitutional disturbance, and death by suffocation if the sufferer be not relieved. If any mucus be coughed up and examined, the parasites (*Strongylus micrurus*) may be discovered. Bronchial irritation occurring in calves during summer or autumn should always be looked upon with suspicion, and its source thoroughly inquired into. If any calves be dead, a careful examination ought to be made, in order that the pathological condition of the lungs should be determined. If none are dead, the mucus from the nose should be examined, when in all probability some of the parasites will be discovered.

*Treatment.*—The calves are to be warmly housed if the nights be cold; the affected animals are upon all occasions to be removed from the healthy; not that the disease is contagious in itself, but that the parasites or their ova, discharged from the sick, are apt to gain access into the bodies of the healthy, and for the same reason the healthy should be removed to fresh pasture and to dry situations, as the fields upon which the disease has prevailed will, for a time at least, be tainted by the parasites and ova.

Inhalations of chlorine or sulphurous acid are recommended. If this be carefully done, the sufferers may be kept surrounded by either of these for about fifteen minutes each day, until the disease disappears; two or three inhalations are generally sufficient. If inhalation be objected to, turpentine may be administered in gruel daily, or, what has succeeded well with

me, from ten to twenty minim doses of Scheele's hydrocyanic acid, with carbonate of soda, and some bitter stomachic, as gentian or chamomile, twice per day. The acid seems not only to have the effect of destroying the parasites, but also of allaying the bronchial irritation in a very short space of time.

The "gapes" in fowls, due to a parasite in the air passages, the *Syngamus trachealis*, and indicated by gaping, gasping for breath, sneezing, and frequent attempts at swallowing, is best treated as recommended by Professor Cobbold and others.

"*First.* When the worm has taken up its abode in the trachea of fowls and other domesticated birds, the simplest plan consists, as Dr. Wiesenthal long ago pointed out, in stripping a feather from the tube to near the narrow end of the shaft, leaving only a few uninjured webs at the tip. The bird being secured, the webbed extremity of the feather is introduced into the windpipe. It is then twisted round a few times and withdrawn, when it will usually happen that several of the worms are found attached. In some instances this plan entirely succeeds. But it is not altogether satisfactory, as it occasionally fails to dislodge all the occupants.

"*Secondly.* The above method is rendered more effectual when the feather is previously steeped in some medicated solution which will destroy the worms. Mr. Bartlett, superintendent of the Zoological Society's Gardens, employs for this purpose salt, or a weak infusion of tobacco; and he informs me that the simple application of turpentine to the throat externally is sufficient to kill the worms. To this plan, however, there is the objection that, unless much care be taken, the bird itself may be injuriously affected by the drugs employed.

"*Thirdly.* The mode of treatment recommended by Mr. Montagu appears worthy of mention, as it proved successful in his hands, although the infested birds were old partridges. One of his birds had died from suffocation; but he tells us that change of food and change of place, together with the infusion of rue and garlic instead of plain water to drink, and chiefly hemp-seed, independent of the green vegetables which the grass plot of the menagerie afforded, recovered the others in a very short time.'

"*Fourthly.* The plan I have here adopted, by way of experiment, of opening the trachea and removing the worms at once. This method is evidently only necessary when the disease has

advanced so far that immediate suffocation becomes inevitable; or it may be resorted to when other methods have failed. In the most far-gone cases, instant relief will follow this operation, since the trachea may with certainty be cleared of all obstructions.

“*Lastly.* The most essential thing to be observed, in view of putting a check upon the future prevalence of the disease, is the *total destruction of the parasites after their removal.*”

*Intra-tracheal injections for hoose in calves.*—Recent experiments have demonstrated that drugs may be injected into the trachea with impunity, and that hoose may be most advantageously treated with remedies administered by this method. Turpentine, prussic acid, carbolic acid, and creosote may be used in this way. The turpentine and creosote should be mixed with either oil, or an emulsion of oil, water, and alkali, as in the following:—℞ Ol. tereb., ʒij.; ol. olivæ, ʒj.; potassæ carb., gr. xx.; aquæ, ʒj. Inject this quantity daily for three days. The injections are given with the hypodermic syringe. Make a small incision through the skin at the site for tracheotomy, pass the needle of the syringe into the trachea between two of the rings, and inject.

Corrosive sublimate, one of the most powerful parasitocides known, might be tried in the form of intra-tracheal injection—gr.  $\frac{1}{6}$  to gr.  $\frac{1}{4}$  in ʒiij. or ʒiv. of water.

#### PARASITIC GASTRIC CATARRH.—(*Gastrorrhœa Parasitica*.)

A disease of the fourth stomach of the sheep, caused by the presence of the nematode worm called the *Strongylus contortus*.

The following is a short history of an outbreak of disease in sheep induced by this parasite, and to which my attention was called by Mr. Connachie, V.S., Selkirk, in March of this year, 1884.

It appears that the owner of the sheep in question had placed his lambs, after weaning time, upon a very luxuriant pasture, but from the nature of the autumn of 1883, which was very wet, the grass became rank and the soil damp, notwithstanding it had been previously well drained both on the surface and underground.

After thriving well for some time, they began to lose flesh very rapidly, and gave other indications of disease; they were then removed to another pasture of good clean grass, and shortly afterwards placed on turnips, oats, and Indian corn. This change seemed to have the desired effect, as they commenced to thrive and lay on flesh; but in the month of February 1884, when nearly fat and fit for the butcher, the now hoggets suddenly

presented signs of disease. Their appetite at first became capricious; they then ceased eating their food altogether, but would devour earth or any rotten or decaying vegetable matter. From this time other and more serious symptoms soon developed themselves, such as great restlessness, lying down and suddenly rising again, pawing with the feet and showing signs of abdominal pain. The bowels now became very irritable, and from being constipated became very loose, the evacuations being like muddy water. At this stage a most unnatural desire for water was evinced, the sheep roaming about in search of it, and when driven away from it, immediately returning to it. Along with this insatiable thirst, there was also an almost constant desire to urinate, the urine passed being as clear as water. The cough now became hacking, but intermittent, each fit of coughing continuing until the animal succeeded in ejecting a quantity of frothy mucus, which relieved it for a time only, as the fits soon returned and became more frequent; during the intervals a flow of watery saliva continued to drivel from the mouth, accompanied by a constant grinding of the teeth, and a continual twitching of the nostrils and upper lips.

During the development of the above symptoms the hogs had most rapidly lost flesh, the adipose and muscular tissues being rapidly atrophied; the abdomen becoming greatly distended, causing the animal to present a most miserable appearance. All the above symptoms increased gradually and steadily, until death closed the scene.

After trying various remedies without success, Mr. Connochie advised the owner, Mr. Elliot, Hollybush, Galashiels, to call me into consultation.

Before seeing the sheep in consultation with Mr. Connochie, one carcase was sent to the College for examination.

In the lungs of this sheep the *Strongylus filaria* was found, but not in great numbers; the parasites were also found in the blood-vessels of the lungs, causing embolism; and in the fourth stomach the *Strongylus contortus* was found in great numbers. After this examination I visited Hollybush, and saw several sheep in an advanced stage of the disease, and, in addition to the above symptoms, furnished by Mr. Connochie, found that the majority presented aggravated symptoms of oedema of the head, affecting the nostrils and interfering with the breathing, and involving the intermaxillary space and inferior cervical region as low down as the breast.

On making a *post mortem* examination of one sheep killed for the purpose it was found that the *Strongylus filaria* was absent, but that the true stomach (abomasum) contained a quantity of a dirty looking fluid, in which the *Strongylus contortus* could be seen wriggling in great numbers.

The parasites were so small as to be seen in the fluid with difficulty by the naked eye, but by gently moving the walls of the viscus, the whole fluid would seem to be one seething, fermenting mass of wriggling, moving worms; whilst the body of the animal was very anæmic, the muscular tissue greatly wasted and very pallid, the fat fairly abundant, but presenting the very white appearance seen in liver-rot and other anæmic diseases.

The *Strongylus contortus* has been studied in Germany by Gerlach particularly, and the following is a brief *resumé* of his conclusions.

The *Strongylus contortus* occurs in the fourth stomach of the sheep and goat, and when present in large numbers causes disease (*Magenwürmerseuche*). At the same time, as was first noticed by Gerlach, the *Strongylus filaria* is usually present in the lungs. In the summer the *Strongylus filaria* is most abundant; in autumn the *Strongylus filaria* and *Strongylus contortus* are almost equally numerous; while in winter the *Strongylus contortus* prevails, and the *Strongylus filaria* has almost disappeared. Hence the stomach disease caused by *Strongylus contortus* breaks out in winter and spring in those lambs which have survived the lung disease of summer and autumn.<sup>1</sup>

These facts agree with what was observed at Hollybush, namely,—1st. The appearance of disease early in the autumn amongst the sheep. This would be due to the lung disease caused by *Strongylus filaria*. 2d. The recovery of many of the sheep affected when the diet was changed, *i.e.*, when removed to a clean pasture, and 3d. The appearance of the disease later on in the season, in February 1884, and resulting from the *Strongylus contortus*, confirmed by the *post mortem* examinations, in one of which only a few *Strongyli filariæ* were found.

From this connection between these worms, Gerlach was led to make the following experiments:—Eggs of *Strongylus filaria* were given to healthy goats and lambs, and, four months after the feeding, *Strongylus contortus* was found to be present. From these experiments Gerlach concluded that *Strongylus contortus*

<sup>1</sup> The *Strongylus contortus* is prevalent in Jamaica in districts where animals are confined to "pond water" and is injurious to cattle and destructive to sheep and goats.

could develop from the eggs of the *Strongylus filaria*, but there is no doubt that his conclusion was erroneous, the two species being absolutely distinct.

The name *Strongylus contortus* occurs first in the writings of Rudolphi, and the species was described from specimens found in the month of November in the fourth stomach of a lamb. Fabricius also found them in the intestine.

*Description.*—The body is cylindrical, red or white, and tapers very gradually towards the anterior extremity, where is the mouth, which is unprovided with papillæ or other appendages, and leads into the muscular pharynx, rather more than 1 mm. in length, in a worm 25 mm. long.

The male is smaller than the female, and is from 10 to 16 mm. in length, and 0·2 mm. in diameter.

The terminal bursa consists of two elongated ovoid lobes, each of which is supported by five ribs, and attached to the left lobe is a smaller one, supported by two ribs. There are two spicules about 0·5 mm. in length, each one tapering to a fine point, which is slightly curved outwards. Under a high power each point is seen to be tipped by a small knob.

The female is from 18·30 mm. in length, and 0·3 mm. in diameter. The body tapers very suddenly to an extremely fine point, and about 3 mm. in front of this is the genital opening, furnished with two papillæ, of which the left is much larger than the right. The uterus is convoluted, hence the name *Contortus*.

*Symptoms.*—The symptoms of the stomach disease caused by the *Strongylus contortus* are, according to Gerlach, not sufficiently marked to allow of a certain diagnosis. The animals are languid and dejected, and tend to become lean and anæmic; the excrement is thin and brown, mixed with slime, or at times with blood. At last cachexia occurs, and death ensues from starvation (*Erschöpfung*). On *post mortem* examination the fourth stomach is found with hundreds of the *Strongylus contortus* covering its surface.

*Treatment.*—Good food is recommended, *e.g.*, roasted malt, seeds, lupine hay or lupine seeds, and the following medicines:—

Chabert's oil,	. . .	1 part.
Oil of turpentine,	. . .	3 parts.

The dose being one tea-spoonful per day.

Kamala has been recommended, and the picrate of potash is highly spoken of by Zurn.

Rabe used picrate of potash in the small dose of 0·12 grm., or about 2 grains per day for three days, and was successful in his treatment. The picrate of potash was given in thick linseed mucilage.

The doses of picrate of potash as given by Zurn are—

For a lamb 0·30 grm. or about 5 grains.

For a sheep 1·25 grms. or about 20 grains.

Divided into two doses per day.

Thymol, which is said to be so specific in human ankylostomiasis, might be tried in these strongyle infections of the stomach and upper part of the intestine.

It is now believed that the ova of these parasites, passed from the body of their host, retained their vitality in damp places only, and that where nice bites of green grass, such as that growing on the sides of open drains and damp spots in pastures otherwise dry, are sources of danger, the parasitic ova becoming ingested with such grasses. The prevention of the disease by the destruction of the worms should be attempted by first digging up, where possible, all such green grasses, and inverting the sods. 2*d.* By a liberal application of salt to the land, and, where possible, an allowance of it to the sheep.

During wet seasons the above precautions are impossible, and all that can be done is to remove to the driest pastures, give corn where possible, and an allowance of common salt in the food.

#### STRONGYLUS TETRACANTHUS.

In March 1873, an Iceland pony rising two years old, which had been under my care for some weeks, died from what appeared to be an exhausting disease, and, as stated in a letter to Dr. Cobbold, there were no very marked signs of the presence of parasites during life. The appetite was good; there was no cough; and we were led to suppose that there were worms by the absence of disease more than anything else. There was gradual emaciation, but no diarrhoea, and at no time were the faeces foetid. As bearing upon the discovery of this parasite, I may state that Dr. Knox, who wrote in the *Edinburgh Medical and Surgical Journal* for 1836, says—"On two occasions my friend Mr. Dick found parts of the large intestines of a horse presenting the following appearance: the gut being simply laid open so as to exhibit the mucous membrane, a number of dark



spots are observed, evidently exterior to the inner membrane." And, further on, speaking of the worms, he adds: "They have no cyst, and vary from the tenth of an inch to a length of at least seven-tenths. They have a digestive tube divided into numerous compartments or sacculated; a mouth unarmed."

Professor Dick's own statements, as communicated by Dr. Knox, are particularly interesting. He says: "I have found

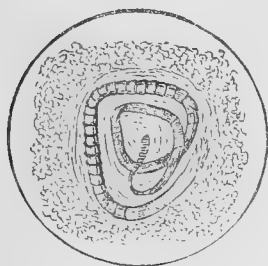


FIG. 86.

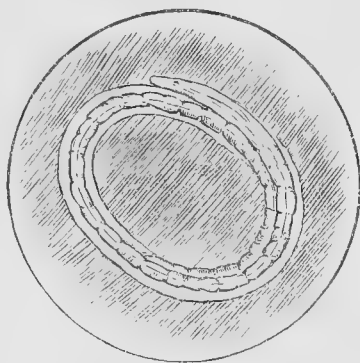


FIG. 87.

Microscopic appearance of *Strongylus tetracanthus* embedded in mucous membrane of Iceland pony, described in the text. These worms were of a bright red colour. (About 100 diameters.)

these worms in several horses, and at different stages of their growth, from the size of a pin-point to that of an inch and a half in length, and in two instances in the blood-vessels.

"They seem almost always to produce a bad form of diarrhœa, and seem to depend on the food or situation in which the animal has been previously kept. The horses I have found affected with these have always been running out previous to their becoming ill."

During the spring of 1874 I had again the opportunity of studying the disease caused by this parasite, or rather parasites, for I am of opinion that there were at least two kinds present in the subjects examined by me, some being embedded in the intestinal walls and others in the intestinal canal, the latter being a reddish-looking worm, very similar to a small earth-worm, from two to three inches in length. Those embedded in the intestinal walls, as well as the appearance of the intestines, are delineated in the above woodcuts.

I may state that all the ponies affected were rising two years old, and that several had died before my opinion was sought. They had been imported from Iceland during the summer and autumn of 1873, and thriven tolerably well until late in the winter. They were at pasture during the whole winter and spring, being allowed hay in addition to what they could graze. There were many older animals amongst them, the lot being a large one, all of which were quite free from the parasites.

The question naturally arises, Are these parasites peculiar to Iceland? and at the first glance, my own experience would compel me to answer in the affirmative; but the observations of others at once point to the conclusion that other than Icelandic ponies are subject to the invasion, and Dr. Cobbold has arranged the following list of those who have written upon the subject:—

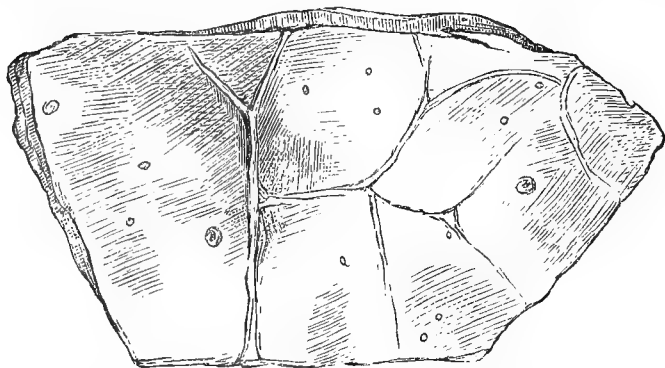


FIG. 88.—Appearance of mucous membrane of colon of Iceland pony, natural size.

“*Dick.*—‘Worms at different stages of growth;’ in MS. to Dr. Knox, 1836.

“*Knox.*—‘Animals similar to *Trichina*;’ in *Edin. Med. and Surg. Journ.* (loc. cit.)

“*Diesing.*—‘*Nematoideum equi caballi*;’ *Syst. Helm.*, vol. ii. 1851.

“*Little.*—‘Extremely small ascarides;’ in a letter to Mr Varnell; *The Veterinarian*, 1864, p. 202.

“*Varnell.*—‘Entozoa in various stages of growth;’ *The Veterinarian*, *ut supra*, p. 265.

“*Williams.*—‘Entozoa from the intestinal walls;’ in MS. to the author, March 13, 1873.

“*Cobbold.*—*Trichonema arcuata*, a new species of nematode (with description as below).

"Body spindle-shaped, more narrowed posteriorly than in front; head truncate, with circular mouth, cup-shaped buccal cavity, and muscular cesophagus; tail of the male drawn out to a long fine point, that of the female being abruptly pointed; reproductive papilla of the female situated at the lower part of the upper third of the body.

"Size.—Males,  $\frac{1}{10}$  to  $\frac{1}{8}$  of an inch in length, by  $\frac{1}{130}$  to  $\frac{1}{120}$  in breadth; the females averaging  $\frac{4}{10}$  of an inch in length by  $\frac{1}{40}$  in breadth."

The opinion that this was a new species of nematode was found to be a mistake, as even Cobbold had referred to it in 1873, and Küchenmeister in 1857.

*Symptoms.*—In the pony under observation in 1873 diarrhœa was absent throughout. Mr. Justus Littler, who was then a student with me, was able, however, to see some points of resemblance in the symptoms to those described by his father in the letter to Mr. Varnell, above mentioned. In the lot observed in 1874, diarrhœa of a fœtid character, along with a more or less rapid emaciation, was present in all the cases, and I conclude that, in conjunction with the ages of the affected animals, diarrhœa and emaciation may be considered as diagnostic symptoms. Four of the 1874 lot were so reduced as to be unable to stand; they were recumbent, very quiet, with pallid mucous membranes; the appetite was, however, good. The question again arises, Was the diarrhœa due to the presence of the larger worms in the intestinal canal? and one is led to conclude that, from the fact that none were discovered in the first case, this symptom arose from this cause, and that so long as the parasites were passive in the intestinal walls they did not cause superaction of the bowels, but as they became matured and burst through the tissue surrounding them, they irritated the intestines and caused purgation. In addition to the dark spots on the mucous membrane of the colon, delineated in the woodcut, the intestinal glands were, in all the cases examined, more or less enlarged, many of them containing pus and what appeared to be the *débris* of the parasites. In every instance the dark spots, which in reality indicated the seat of the worms, were confined to the colon. An experiment was made with the flesh of the first pony upon some fowls, and, as stated by me in my letter to Dr. Cobbold, two of them died, with the livers and other viscera filled with parasitic ova and embryos.

Mr. Cawthron of Hadlow has found these parasites enveloped in cysts of vegetable *débris*.

*Treatment*.—In the cases treated in 1874 we were successful in every instance by administering two-drachm doses of the oil of turpentine night and morning, along with eggs and milk well beaten up. The eggs and milk were administered, not only for the reason that they form a good vehicle for turpentine, but to afford nourishment and support to the debilitated animals.

This is a very interesting subject, requiring further investigation, and it may turn out that the larger worms are a more mature form of the smaller specimens, or that they are a distinct form altogether.

#### TRICHINOSIS.

The *Trichina spiralis* is a minute round-worm, measuring about  $\frac{1}{35}$  of an inch in length, originally found in the muscular tissue of pigs, cats, dogs, badgers, etc., and transmissible by ingestion of trichinosed flesh to other animals, and to man; but, according to some observers, carnivorous birds are exempt from the invasion of the parasite. The anterior extremity of the worm is rather pointed, its posterior thick and rounded; has immature sexual organs, and lies coiled up in an oval cyst. The cyst, which measures about  $\frac{1}{70}$  of an inch in length, appears to be no essential part of the worm, but forms around it after it has taken up its location; the walls of the cyst are laminated, transparent, and thick, generally studded externally with calcareous matter.

The trichina cysts occupy the striated muscular tissue, and in some specimens it has been estimated that each cubic inch of the striped muscles of an infected hog may contain from thirteen to thirty-five thousand worms. The cysts appear in the muscles as minute white grains, visible to the naked eye, and with their long diameter corresponding to the direction of the muscular fibres. In a cat experimented upon by Leuckart each ounce of muscle was calculated to contain 325,000 trichinæ; and Dr. Cobbold estimates that a man of medium bulk may easily harbour 20,000,000. Dr. Bellfield and Mr. Atwood, at Chicago, fed a rat, weighing two ounces, with infected pork in small quantities every two or three days for six weeks. No impairment in the health of the rat resulted; it was then killed, and

its body was found swarming with living trichinæ; the observers estimated there were no less than 100,000 in the whole carcase. They also found that rats may be fed occasionally with small numbers of trichinæ without in any way disturbing the health; and the inference drawn from these experiments has been, that any animal or man may take live trichinæ in small numbers occasionally without injury. These observers further believe that many more human beings than have been hitherto supposed are infected with trichinæ; indeed that the majority of us are carrying these worms in our muscles. So sure have they felt of this fact, that one of them ate twelve living trichinæ on 20th November 1878, and after more than three weeks had elapsed—when the case was reported—not the slightest effect had been experienced. These gentlemen also discovered that small portions of sulphurous acid dissolved in the brine in which hams are pickled will kill the trichinæ. The per-centage of acid was not then fully determined, but the amount is so small that it is no detriment to the meat for commercial purposes.

This worm was first discovered by Professor Owen in 1835 in a piece of a man's muscle, which presented a peculiarly speckled appearance. These specks were found by Professor Owen—as had been previously shown in similar instances by Tiedman and Mr. Hilton—to consist of minute encysted entozoa, which he named *Trichina spiralis* owing to its hair-like and spirally coiled form; but until 1860 it was only known as a pathological curiosity. At that date, however, Zenker of Dresden showed that, however harmless the encysted parasite might be, the gravest symptoms, and even death itself, might be caused after its reception into the bowels, during the process of its reproduction which then ensued, and during that of the migration thence of the young trichinæ into the voluntary muscles.

It is not yet determined how long the larval trichinæ retain their vitality, but there is no doubt that they may live encysted in the muscular tissue for many years, and retain life after the death and putrefaction of their host. They do, however, perish *in situ* sooner or later, and usually undergo calcareous change.

When the trichinæ capsules are swallowed, they are dissolved by the gastric juice, and the contained parasites set free; they then rapidly undergo development, and attain sexual maturity,

the female acquiring a length of  $\frac{1}{8}$  inch, the male  $\frac{1}{16}$ . The males are filled with sperm-corpuscles, and the females densely stocked with ova, which are hatched within the uterus, from whence the living embryos escape into the intestinal canal of the host, and at once commence active migration. They first attach themselves to the intestinal mucous membrane, eat through the intestinal walls, and find their way to the small vessels and lymphatics of the bowels, from which they are conveyed by the blood stream to all parts of the body. They have been found during this period in almost every part of the organism; in the intestinal walls; in the abdominal cavity, in the mesentery and mesenteric glands, in the connective tissue, and, in an as yet unencapsuled condition, in the muscular tissue itself.

The immature trichinæ taken into the stomach become mature on the second day; on the sixth and following days, up to the end of the second or even third week, the embryos are born, and commence operations, and probably reach their destination in a week or two, and by the end of a month or a little more have come to the conclusion of their labours. Although the trichinæ fix their abode in the striped muscular tissue, they are rarely if ever found in that of the heart, but are often specially abundant in the muscles of the larynx.

The experiments of Dr. Bellfield and Mr. Atwood support the conclusions of previous observers, that the manifestations of disease in animals experimentally infected vary according to the number of trichinæ introduced; if these have been few in number, no ill consequences have resulted, but when more numerous, the disease has been of an aggravated or even fatal character.

*Symptoms.*—The symptoms of trichinosis in man, as concisely described in Tanner's *Practice of Medicine*, are as follows:—"The *symptoms* of trichiniasis vary in degree, being mild or severe according as only a few or many of the worms have been swallowed, as well as in proportion to the number of the progeny and the extent of their migrations. Thus, Dr. Althaus remarks that in the epidemic of Burg, near Magdeburg, a woman who had eaten a quantity of raw pork with bread, fell ill and died; her child, who had sucked a spoon used by the mother, suffered slightly and recovered.—According to the accounts given by most authors, the earliest symptoms are loss of appetite and

general malaise; to which succeed nausea and retching, prostration, diarrhoea, a sense of thorough indisposition, and a painful stiffness about the neck and arms and legs. This pain is due to the immigration of the young trichinæ into the muscles; and it is accompanied with high fever, and an œdematous swelling about the eyelids and face. The pulse is frequent, and there are copious offensive perspirations; but although the temperature of the body is raised it does not reach the same height as in typhus and typhoid fever. For some days the stiffness of the limbs continues to increase; while all the muscles seem to be painful and swollen and very sensitive to the touch. The movements of the intercostal muscles in respiration are attended with suffering, so that repose is impossible; while there will be troublesome hiccup if the diaphragm be invaded, with hoarseness and loss of voice where the laryngeal muscles get inhabited. Neuralgia of a very severe description, in the cœliac and mesenteric plexuses, has likewise been present in certain cases. When a large quantity of trichinous meat has been eaten, so that the immigration of the trichinæ into the muscles is great, the patient may lie almost paralyzed in a state of great exhaustion. The facial œdema generally lasts about a week, its disappearance being followed by swelling of the feet and legs, and ultimately of the trunk. There is no effusion, however, into any of the cavities; nor does the urine become albuminous, although it is always lessened in quantity and may be loaded with urates. About the beginning of the fourth week the patient is in a pitiable condition. The pulse and respirations are very frequent, the tongue is red and dry, the pain is severe, the sweating is profuse, the mouth can scarcely be opened, no sleep can be obtained, and there is great anxiety or delirium; death not unfrequently occurring with all the symptoms of profound exhaustion. Such complications as pneumonia, peritonitis, and pleurisy with effusion, are not uncommon. In favourable cases, however, the pain and swelling and diarrhoea abate; the oppression of the chest passes off; sleep is obtained; a desire for nourishing food is evinced; the power of the limbs is regained; and there is only left great anæmia, with a falling off of the hair, &c. The parasites have taken up their abode in the muscles, and have fortunately become encysted."

It appears that the symptoms are much more severe in the

human being than in the lower animals; and Dr. Cobbold mentions that a pig experimented upon at the London Veterinary College, and in which it was calculated that about sixteen millions of encysted trichinæ had been developed, manifested no symptoms of suffering; other pigs, however, showed much general disturbance and suffering, arising from the irritation of the worms in the intestines, and during their passage into the muscular tissues. The irritation of the alimentary canal, which lessens towards the end of the first week after pigs have swallowed trichinæ, is denoted by loss of appetite, vomiting, colic, tympanitis, and diarrhœa; dulness, arching of the back whilst standing; and pigs incline to lie down and hide themselves in their litter in this as in many other diseases. These symptoms, with the exception of the diarrhœa, generally disappear in from six to eight days. When the trichinæ have been numerous, the membranous passage of a great number of them have, in the experience of Leuckart, induced in some a fatal peritonitis; and in others, a form of enteritis, with ejection of false membrane.

"The muscular symptoms appear towards the termination of the second or during the third week after the ingestion of the trichinosed flesh; while the intestinal phenomena become less marked, and the fever increases in intensity. At this stage the patient is often lying; in walking, it carries its back raised and the limbs stiff, and manifests signs of pain; mastication, opening the mouth, and swallowing are difficult; the voice is harsh, husky, and weak; the respirations laboured and loud; cutaneous œdema appears in different places, together with the intense pruritis already alluded to; the conjunctival membrane is most frequently infected, and emaciation rapidly sets in. If the number of migratory trichinæ is very great, death may be the result, though this is somewhat rare in the pig. Usually the different symptoms disappear gradually, and it is only in very debilitated animals that convalescence is slow. The presence of trichinæ in the muscles does not appear to exert any subsequent influence on the development or fattening of the animals which harbour these strange creatures; on the contrary, it has been stated that they fatten more readily than before they were infested."—(FLEMING'S *Sanitary Science and Police*.) The determining test, however, is the discovery of the parasites in the intestinal discharges, or in fragments of muscular tissue extracted by a harpoon.



The vitality of the trichinæ is not destroyed unless the meat in which they are contained is exposed to the influence of the heat of boiling water for a sufficient time to insure that every particle of it has been acted upon by that degree of heat. The mere toasting of ham or bacon is insufficient to destroy the worms, and smoked ham and German sausages are, unless well cooked, sources of danger. Many remedies have been suggested for the destruction of the trichinæ in the treatment of the disease, more particularly picric acid, picro-nitrate of potash and benzole, carbolic acid, sulpho-carbolate of soda, &c., but none of these have been proved to have any effect; indeed, in trichinous pork of a pig killed with picric acid, the worms were found alive by W. Müller of Homburg.

## CHAPTER LXXXV.

### PARASITIC DISEASES—*continued*.

#### TREMATODA.

##### ROT IN SHEEP.

ROT is a disease of low lands, marshy grounds, and wet seasons, and observers are agreed that the flooding of pastures suffices to taint them for a season, owing to the dissemination of the *Distomata*; for it is pretty satisfactorily proved that the ova of the fluke pass out through the intestines of the sheep, and fall on the pastures. The ova hatch in moist places, and become transformed into ciliated embryos, which, when set free, swim rapidly in the water, and thus spread over pastures in wet seasons.

Remarkable outbreaks of this disease have occurred in England. Professor Simonds, in his essay, records the outbreaks of 1809, 1816, 1824, 1830, 1853, and 1860. "In the outbreak of 1853-54 many thousands of sheep were swept away, and not only in undrained districts, but also in others of a more healthy character. But since 1830, however, no outbreak can at all be compared to the one of the autumn of 1860. Speaking in general terms, it may be affirmed that all the western and southern counties of England, together with several of the eastern and midland, suffered to a ruinous extent. As in former years so in this, the attacks of the disease were due to an excess and long continuance of wet weather. 1860 will long be remembered by agriculturists, not only as producing the rot among sheep, but likewise for its baneful effects on the root crops, as also on the hay and corn harvests." 1872 again was a wet year, and rot was very prevalent, appearing on land that was considered sound during ordinary seasons.

In conjunction with the *Distomata* in the liver, it is not an unusual thing to find other parasites infesting the bodies of sheep. Strongyles in the abomasum, intestines, and lungs, cysticerci, &c.—indeed symptoms similar to those of rot—are sometimes present independent of the fluke, and this circumstance has led some erroneously to conclude that the debilitated condition of the sheep renders it a favourable habitat for the parasite; that, in fact, the mal-condition induced by damp food is the cause of the disease and not the effect, hence it is termed watery cachexia, *cachexia aquosa*, by some writers.

It is important that this disease be distinguished in its earliest stages, and in order to enable the sheep-farmer to do this, the following rules are given by Mr. Beattie in the *Transactions of the Highland and Agricultural Society of Scotland*, vol. iii., 1807:—

“The first thing to be observed is in the spring, when they are dropping their lambs. A sound ewe in good order drops a lamb covered with a thick and yellow slime, which the ewe licks off it, and the rule is, the sounder and the higher condition the ewe is in, the darker and thicker will be the slime; but when they observe a ewe drop a lamb covered with thin watery bubbles, and very white, they note her down as unsound.

“About the month of September, when they intend to dispose of their draught ewes, they put all their sheep into a fold, and draw them by the hand; that is, they catch them all, viz., the ewes they design to sell any of, and clapping their hand upon the small of the back, they rub the flesh backwards and forwards betwixt their fingers and thumb and the ends of the short ribs. If the flesh is solid and firm, they consider her as sound; if they find it soft and flabby, and if, when they rub it against the short ribs, it ripples, as we term it—that is, a sort of crackling is perceived—as if there was water or blubber in it, they are certain she is unsound. This is the most certain of all symptoms, but is not to be discerned with any degree of certainty but by an experienced hand; for although, as I have here related it, it seems a very simple affair, and easily acquired, yet it is well known that many shepherds, who have followed sheep all their lives, never arrive at anything like certainty in judging by the hand, whilst men of superior skill will seldom be mistaken, and will draw by no other rule. Yet still it must be acknow-

ledged that the seeds of this disease will sometimes lie so occult as to baffle all skill, and that no man can, with absolute certainty, draw a flock tainted with the rot. There is another method, to which men of inferior skill resort, which is more easily acquired. They take the sheep's head between their hands, and press down the eyelids; they thereby make the sheep turn its eyeball, so that they get a view of the vessels in which the eyeball rolls. If these are thin, red, and free of matter, they consider the sheep as sound; but if they are thick, of a dead white colour, and seem as if there was some white matter in them, they are confident she is rotten. This is a pretty general rule, and easily discerned; but I think it is not so certain as when they are judged by the back; for in firm heathy lands the eye of a sheep is far redder than it is in sheep upon grassy lands; and in some boggy lands, the eye is never very red, be the sheep ever so sound, so that there you cannot so well judge by the eye; but when you see the eye of a sheep a good deal whiter and thicker, and more matter in it (I mean the vessels in which the eyeball rolls) than the run of the flock amongst which it feeds, you have reason to suspect it is not sound."

In some instances the progress of the rot is very rapid, but usually it is slow and insidious. At first the affected animals appear to thrive very fast, but inactivity and dulness supervene; the mucous membranes become pallid, the flesh wastes, the general surface of the skin loses its ruddy colour, becomes dry and devoid of that oily condition which is natural to the fleece of the sheep. As the disease progresses, the flanks become hollow, the back rigid, weak and tender about the loins, as evinced by wincing when this part is pressed by the hand, and the spine sticks out prominently; the fleece drops off in patches, the belly enlarges, the eyes become yellow, and dropsical swellings appear in different parts of the body, particularly around the throat. There is often much thirst, depravity of the appetite, diarrhoea, general stupor, the pulse is weak, the heart's action tumultuous, and anæmic murmurs are heard. As demonstrated by Delafond and others, the blood is deficient in albumen, thin, watery, and on this account the serum transudes through the walls of the vessels, collects in the loose areolar tissue of the depending parts of the body and in the cavities, constituting the condition of dropsy which is seen in rot.

Professor Simonds states that a dry scaly state of the skin on the inner parts of the thighs, particularly where it is uncovered with either wool or hair, is early recognised, and that an examination of the eye will materially assist in determining the question of disease. "If the lids are everted, the *membrana nictitans* being pressed forward, it will be found that in the early stages of the malady, and especially if the animal has been excited by being driven a short distance, the vessels of the conjunctiva are tinged with a pale or yellowish-coloured blood, and that the whole part has a moist or watery appearance. Later on the same vessels are blanched, and scarcely to be recognised, excepting perhaps one or two, which present a similar watery condition, or are turgid with dark-coloured blood." In some cases these symptoms are complicated by others, induced by strongyles in the air passages and alimentary canal.

#### PATHOLOGICAL ANATOMY.

The tissues of the body are generally wasted, flabby, pale or yellow, and watery; there is an absence of the firmness and colour of healthy mutton. The peritoneal cavity contains a more or less abundant quantity of serum, which may be of a clear straw colour, or more or less yellow, in which occasionally fragments of lymph and false membranes are floating; the digestive organs are remarkably blanched; the liver is hard, scirrhus, irregularly knotted on its surface and margins, and sometimes united by false membranes to the surrounding organs. In colour it is either a dirty chocolate brown, deeper in some parts than others, or has a yellowish tint, intermixed with pale yellow spots. Flukes are found in the bile ducts, which are filled with a dark thick secretion; on further examination the ducts are found sacculated at various points, the distended portions generally containing many flukes massed together. The canal walls are much thickened in some places, and coated with calcareous matter on their internal surface. Professor Simonds mentions a case where a concretion was found as large as a hen's egg, which, when broken up, was found to contain about a dozen dead flukes. He also states that "the coats of the *ductus hepaticus*, as also of the *ductus communis choledicus*, are not unfrequently so thick as to be upwards of ten times their normal

substance, and likewise as hard as to approach the nature of cartilage."

Respecting the number of the flukes, the greatest variation exists. Dr Cobbold says—"The presence of a few flukes is totally insufficient to cause death; consequently, when a sheep dies from rot or is killed at a time when the disease has seriously impoverished the animal, then we are sure to find the organ occupied by many dozen, many score, or even several hundred flukes. Thus from a single liver Bidlow obtained 800, Leuwenhoeck about 900, and Dupuy upwards of 1000 specimens. Even the occurrence of large numbers only destroys the animal by slow degrees, and possibly without producing much physical suffering, excepting perhaps in the later stages. Associated with the above described appearances, one not unfrequently finds a few flukes in the intestinal canal, whilst a still more interesting pathological feature is seen in the fact that the bile contained in the liver ducts is loaded with flukes' eggs. In some cases there cannot be less than tens or even hundreds of thousands. Not a few may also be found in the intestinal canal, and in the excreta about to be voided." And lastly, says the same author, "it need scarcely be added that it is by no means unfrequent to find one or even several other kinds of entozoa coexisting with the fasciola in the same sheep, the most common form being that of the larval echinococcus."

#### TREATMENT.

All observers agree that the growth and development of the fluke is impossible on dry land, and that the disease is unheard of on dry pastures except during wet seasons, and as the "rot," once established, is difficult to overcome, it behoves the flock-master to pay every attention to its prevention. For this purpose it is necessary that unsound sheep pastures should, after being drained, for a time at least, be applied to other purposes.

During moist seasons, and when there is a fear of the disease appearing on sound pastures, the natural food should be supplemented by cakes, corn, beans, or other nutritious diet, in addition to common salt and the sulphate of iron.

As to the beneficial effect of common salt, all writers are agreed that it prevents the development of the parasites, for it

is tolerably certain, as mentioned by Dr. Cobbold, that the larvæ of the *Fasciola hepatica* exists only in the bodies of fresh-water snails. Seeing this is the case, and that salt marshes are exempt from the invasion of the fasciola, would not the application of common salt to the land be worthy of a trial?

For sheep that are actually affected but little can be done, at least in the way of a permanent cure. If the disease be slight, its progress may be retarded, and the animals made fit for the butcher, by removing them to a dry pasture, supplying them liberally with food of a highly nutritious nature, and administering salines and tonics.

#### BILHARZIA.

The wasting symptoms, œdema, and anæmia described are those noticed in animals in which bilharzia and their eggs were found. The treatment and hygienic principles adopted in fluke disease have also been recommended for schistosomum infection.

## CHAPTER LXXXVI.

### PARASITIC DISEASES—*continued*.

#### CESTODA.

THE most important disease of stock caused by this order of parasites is that variously termed

#### STURDY, TURNSICK, OR GID.

This is a species of vertigo induced by the *Cœnurus cerebralis*, which is the hydatid or larval form of the tape-worm, called the *Tænia cœnurus*. Many conclusive experiments have been made in this and other countries, recorded in works on helminthology, which proves that the mature *Tænia cœnurus* infests the intestines of the dog, from which perfect segments are expelled. These segments, falling on the pastures where sheep are feeding, are swallowed along with the herbage, and, as explained by Dr. Cobbold, the six-hooked embryos, escaped from the ova contained in the segments, commence their wandering in the tissues of the higher animal, and, by virtue of their selective capacity, escape from the blood-vessels of the brain, and bore their way to their final resting-place in that organ. Even when the brain is reached, it by no means follows that every embryo attains its full growth. Indeed, it is only usual for one to flourish, sometimes two of equal size, but seldom more. It has, however, been observed that when one hydatid cyst has been removed, that another has developed in a different part of the brain. This proves that the brain serves as a proper habitat to one or at most to two hydatids at a time.

This disease attacks cattle as well as sheep, but in this country it seldom attacks any but sheep, its victims being lambs under



one year old, sheep above two years old being rarely affected. It prevails to a great extent on unenclosed lands, and for the reason that shepherds have to be assisted by dogs, whilst on enclosed pastures, and where sheep are unattended by dogs, the disease is scarcely known.

*Symptoms.*—The usual form of sturdy is that due to the location of a hydatid in one of the cerebral hemispheres. The sheep so affected, when caused to move, turns from right to left, or left to right, as the case may be; but when the parasite is lodged between the hemispheres, the animal steps high, and goes forward in a straight line; the head is then carried upwards, and there may be a varying degree of amaurosis in one or both eyes. Sometimes the animal becomes both blind and deaf, and is unable to follow its companions. When the hydatid is lodged in the cerebellum, the animal's movements are performed without control. The head is elevated; the limbs are moved with difficulty and automatically; one or two steps are taken forward, when the animal starts with a bound, but immediately falls, and is unable to rise for a time.

When the animal is first affected the symptoms are generally severe, from the congestion and irritation; as the contents of the skull adapt themselves to the hydatid, the brain symptoms subside more or less; but as the parasite grows, the symptoms become more severe, until paralysis is induced, and the animal can no longer stand.

As many tumours and hydatids of different species are found in the brain which do not induce symptoms of sturdy, Davaine believes that the nervous substance is irritated by the heads of the parasite, which protrude from the bladder, and by means of their hooks and sucking discs penetrate the brain substance nearly two lines in depth. As the cœnurus grows, the animal becomes more and more emaciated, and death occurs, unless the sheep be relieved naturally or artificially. The natural method is by the bones of the skull becoming absorbed, the skin accidentally broken, and evacuation of the hydatid. This is, however, very rare.

The bones of the skull, however, are generally softened, and if in about three weeks after the first manifestation of the symptoms the head be pressed with the thumb, a remarkable degree of softening will be found at one part of it, as if the skull

were wanting in that particular spot. Into this softened spot a trocar and cannula are introduced, and after the trocar is withdrawn—leaving in the cannula—a syringe is applied to the latter, and the bladder extracted.

“The prevention of the hydatid disease,” says a writer in the *Agricultural Gazette*, “must begin at its source, and the first step is to expel the tape-worm from the intestines of the dog or dogs which are employed in the farm. It may be objected that this measure will be applied too late to remedy the mischief if the existence of the tape-worm is not suspected until some of the flock become affected with “gid;” but in fact the existence of tape-worm should always be suspected, and dogs should be treated periodically with areca nut as a measure of precaution. A dose of half a drachm to two drachms of the grated nut may be given now and then with safety, even when the tape-worm segments are observed, and if the remedy is not followed by the expulsion of a worm, the animal may be deemed secure. A second important means of prevention is to prevent the infection of dogs by destroying bladder worms immediately on their removal, instead of throwing them either to the dogs or in places where the animals can easily discover them. If this plan were universally adopted cystic entozoa would soon be diminished in number, and one source of loss to the stock-owner be averted. Sheep suffer from the entrance of embryos, which are expelled from the intestines of the dogs. Dogs become infested with the tape-worm in consequence of introduction into their digestive organs of the larval forms of the parasites, which exist in the brain of the sheep, being given to them, or placed within their reach. This interchange can be prevented by the exercise of a little common care.

“Sheep which are pastured on common lands, or fields through which a right of way exists, are exposed to risks of various kinds from which animals in isolated positions are exempt, and under such circumstances it is impossible to apply any effectual measures of prevention. In the case of “gid” some amount of danger is incurred from wild animals; foxes, for instance, may harbour the *Tænia cœnurus*, and a few sheep may here and there become infested with bladder worms from eating the eggs expelled from the intestines of those animals, but these causes of infection are occasional and remote, and do not materially affect

the main question. Of the method of prevention, if the *Tænia œenurus* were to be expelled from every dog in the country, and effectually destroyed, the disease "gid," among sheep, would be very rare indeed.

"In reference to the treatment of the disease we have nothing to urge. Puncture or removal of the cyst in many cases alleviates the distress which the animal suffers, but permanent cure is rare, and, knowing this, the farmer wisely consigns the animal to the butcher. He should, however, always bargain for the return of the head, which contains the parasite, in order that he may burn, bury, or otherwise destroy it, and thus render thousands of embryos inert."

The various other diseases caused by *tæniæ*, in different stages of development, and other parasites, almost too numerous to mention, are not intended to be described in the present work; helminthology being a specialty upon which many works have been written. In connection with this I may refer to one small work, namely, *The Internal Parasites of our Domesticated Animals*, by Dr. Cobbold, as being a book which deals exclusively with the entozoa of interest to the veterinarian. Under the able direction of Dr. Tommasi, an Italian edition of this manual has been published at Florence. The reader is recommended to Raillet and also to Macqueen's translation of Neumann's parasites.

I may, however, state that I have found areca-nut, with the etherial extract of the male shield fern, to be the most certain remedy for the expulsion of tape-worms, a tabular arrangement of which is given on the following page.

The drawings of the parasites have been mainly taken from Zurn, also from Heller, Krabbe, Leuckart, Cobbold, Kuchenmeister, and others.

## TABULAR ARRANGEMENT OF ENTOZOA.

## N. O. NEMATODA.

*Genus Ascaris.*

- (a.) *A. megalcephala* of horse and ass ; found in the small intestine, occasionally passing from the duodenum to the gall ducts.
- (b.) *A. lumbricoides* of man, pig, cattle ; found in small intestine. The *Ascaris* of the pig is sometimes reckoned as a different species, viz., *A. suilla*.
- (c.) *A. mystax* of cat and dog ; rarely in man. Found in the small intestine. The *Ascaris* of the dog is sometimes reckoned as a different species, viz., *A. marginata*.

*Genus Eustrongylus.*

- (a.) *E. gigas* of dog, horse, cattle, and very rarely man ; found in the kidneys, bladder, and areolar tissue, beneath the peritoneum ; sometimes free in the peritoneal cavity ; also in the heart of the dog.

*Genus Filaria.*

- Species (a.) *F. lachrymalis* of horse and ox ; found in the lachrymal ducts.
- „ (b.) *F. papillosa* of the horse, ox, and ass ; found in the globe of the eye, said to be in the anterior chamber, but usually in a cyst within the cornea, also in the peritoneal and thoracic cavities, in the diaphragm and abdominal muscles, and in the arachnoid membrane of the brain.
- „ (c.) *F. immitis* of the dog ; found in heart, pulmonary arteries, and blood, hence spoken of as *Hæmatozoa*.
- „ (d.) *F. trispinulosa* of the dog ; found by Gescheidt in the capsule of the crystalline lens.

*Genus Spiroptera (Filaria of some authors).*

- Species (a.) *S. megastoma* of the horse ; found in tumours usually at the cardiac end, more rarely at the pyloric end, of stomach ; when these tumours are pressed, pus and bundles of the worms are obtained. *S. microstoma* of the stomach of the horse is considered as a large variety of *S. megastoma*, but never found inside the tumours.
- „ (b.) *S. sanguinolinta* of the dog and wolf ; found in tumours of the œsophagus and stomach.

*Species* (c.) *S. strongylina* of the pig, &c. ; found in the stomach.

„ (d.) *S. scutata œsophagea bovis* of ox, also described in the horse ; found in the mucous membrane of œsophagus. Worms supposed to be the same are found in tongue of pig and œsophagus of sheep.

„ (e.) *S. hamulosa* of the common fowl.

„ (f.) *S. cincinnata* (*Onchocerca reticulata*) of horse ; found in the foot ligaments wound round the elastic fibres.

Larval forms of nematodes are found in tumours in the sub-mucous layers of the wall of the alimentary canal.

#### *Genus Oxyuris.*

*Species* (a.) *O. curvula* of the horse and ass ; found very frequently in the cæcum and colon.

„ (b.) *O. vermicularis* of man ; found in the large intestine and rectum.

#### *Genus Dochmius* (*Strongylus* of some authors).

*Species* (a.) *D. hypostomus* of the sheep, goat, and other ruminants ; found in the intestine.

„ (b.) *D. tubæformis* of the cat ; found in the duodenum.

„ (c.) *D. trigonocephalus* of the dog ; found in the stomach and small intestine. A variety is declared to exist in the right side of the heart.

„ (d.) *D. cernuus* of sheep ; found in small and large intestine.

„ (e.) *D. duodenalis* (*Anchylostoma duodenale*) of man ; found in duodenum

#### *Genus Schlerostomum.*

*Species* (a.) *Schlerostomum armatus* of horse, or *equinum* ; sexually mature adults found in cæcum and colon, seldom in the duodenum, in the pancreas and tunica vaginalis of testis ; the larval forms in aneurisms in the intestinal arteries, &c. ; the eggs come out with the excrements and develop free living larval forms in water ; the free living larvæ get into alimentary canal with the water, and thence into the blood-vessels, where they cause aneurisms ; becoming sexually mature they leave the aneurisms, and bore their way into the alimentary canal, where they copulate.

„ (b.) *Schlerostomum tetracanthus* of horse ; found in small intestine and cæcum ; the larval forms are free under the epithelium or enclosed in capsules in the mucous

membrane of the large intestine, where they attain sexual maturity, and then leave the mucous membrane and enter the alimentary canal.

*Species (c.) S. dentatus* of pig; found in large intestine.

„ (d.) *Syngamus trachealis*, from the trachea and bronchial tubes of fowls.

„ (e.) *S. radiatus* of the ox, and several other ruminants. From the small intestine and colon.

„ (f.) *S. venulosus* of the goat; found in the small intestine.

„ (g.) *S. micrurus* of cattle, horse, and ass; found in aneurisms of the arteries of the cow, and in the trachea and bronchial tubes. In the tough, yellow mucus of the air passages and bronchi they are to be found in countless numbers.

*Species (h.) S. filaria* of sheep and goat, camel, &c.; found in the trachea, bronchial tubes, and lung-parenchyma. The ripe worms are coughed up and die. From the bodies of the dead females the embryos come forth. When they get into water they develop further, and live as free nematodes (or possibly as parasites on an intermediate host). These gain access to the stomach of the sheep with the water, wander into the lungs, and encyst in the parenchyma. When mature they leave the parenchyma for the bronchi, The forms described as *Nematoideum ovis pulmonale* are considered as embryos of *S. filaria*.

„ (i.) *S. paradoxus* of the pig; found in the trachea and bronchial tubes.

„ (k.) *S. filicollis* of the sheep; met with in the small intestine.

„ (l.) *S. ventricosus* of cattle; found in small intestine.

„ (m.) *S. inflatus* of cattle, rare; found in colon.

„ (n.) *S. contortus* of sheep and goat; found in the abomasum, usually associated with *S. filaria*.

*Stephanurus dentatus* of pig; found in or about the kidneys. The kidney-worm of the Americans; Leuckart's *Sclerostoma pinguiicola*.

#### Genus *Trichina*.

*Species (a.) T. spiralis*; sexually mature adults in alimentary canal producing living young, which bore their way into the muscles, and encyst, forming the immature larval *T. spiralis* or Muscle *Trichina*; found in man, pig, ox, rabbit, rat, &c.

*Genus Trichocephalus.*

*Species* (a.) *T. dispar* of man.

„ (b.) *T. affinis* of sheep and goat; rare in cattle; found in  
cæcum.

„ (c.) *T. depressiusculus* of the dog; found in the cæcum.

„ (d.) *T. crenatus* of the pig and wild boar; found in the large  
intestine.

## N.O. TREMATODA.

*Genus Distoma (Fasciola of some authors).*

- (1.) *Distoma hepaticum*.—Leaf-like form, with a small sucker round the mouth, and a little behind this a ventral sucker, also small. Habitat, gall ducts and gall bladder (usually only in spring) of sheep, cattle, goat, and pig. Rarely in horse, ass, cat, and very rare in man.
- (2.) *Distoma*, or *Dicrocoelium lanceolatum*.—Lance-like form, with a tolerably large ventral sucker. Habitat, gall ducts and gall bladder of sheep, cattle, goat, pig. Wandering specimens also in blood-vessels and heart.
- (3.) *Distoma campanulatum*, according to Ercolani, occurs in the liver of the dog.
- (4.) *Distoma conjunctum*, found in the bile ducts of Indian dogs.
- (5.) *Distoma* species have been found in muscles of pig, more particularly in the diaphragm.

*Genus Amphistoma.*

- (1.) *Amphistoma conicum*, cone-shaped, thick posteriorly, with a large sucker at the posterior end. Habitat, paunch of cattle.
- (2.) *Amphistoma truncatum* of cat.

*Genus Hemistoma—Holostoma.*

- (1.) *Hemistoma alatum* of dog, wolf, and fox; found in small intestine.
- (2.) *Hemistoma cordatum* of cat.

*Genus Gastrodiscus.*

- (1.) *Gastrodiscus polymastos*, a disc-shaped trematode found by Dr. Sonsino in Egyptian horses.

## N. O. CESTODA.

(Fam. 1.) *Tæniadæ*.

MATURE WORMS.	HABITAT.	LARVAL, CYSTIC, OR HYDATID STAGE.	COMMON HABITAT.
1. <i>Tænia medio-canellata</i> .	Intestines of man.	<i>Cysticercus bovis</i> .	Muscles of horned cattle.
2. <i>Tænia solium</i> -	Do. of do.	<i>Cysticercus cellulosæ</i> .	Muscles of pigs.
3. <i>Tænia serrata</i> -	Do. of dog.	<i>Cysticercus pisiformis</i> .	Entrails of hares and rabbits.
4. <i>Diplydium caninum</i> .	Do. of dog, cat, and man.	<i>Cryptocystes</i> in the <i>Trichodectes latus</i> and <i>Pulex serraticeps</i> .	Body of dog louse — <i>Trichodectes latus</i> .
5. <i>Tænia cœnurus</i>	Intestines of dog.	<i>Cœnurus cerebralis</i> .	Brain of herbivora.
6. <i>Tænia echinococcus</i> or <i>hydatid tape-worm</i> .	Do. of do.	<i>Echinococcus veterinorum</i> .	So-called bladder worms, in liver, heart, lungs, bones, &c.
7. <i>Tænia marginata</i> .	Do. of do., wolf, &c.	<i>Cysticercus tenuicollis</i> , or slender-necked <i>hydatid</i> .	Liver, walls of bile ducts, mesentery, pleura, pericardium, diaphragm, &c., of sheep and pigs.
8. <i>Tænia crassicolis</i> .	Do. of cat.	<i>Cysticercus fasciolaris</i> .	Liver of rat and mouse.

*Tænia* whose *Cystic* forms are unknown.

1. *Tænia nana* of man.
2. *Tænia expansa* of ox, sheep, gazelle, chamois, &c.
3. *Tænia denticulata* of ox (France and Germany).
4. *Tænia plicata* of small intestines and stomach of horse.
5. *Tænia mamillana*: large intestines of horse.
6. *Tænia perfoliata*: cæcum, and sometimes small intestines of horse.
7. *Tænia infundibuliformis*,
8. *Tænia proglotina*,
9. *Tænia crassula*,
10. *Tænia malleus*,
11. *Tænia lanceolata*,
12. *Tænia setigera*,
13. *Tænia sinuosa*,
14. *Tænia fasciata*,

Common fowl, water fowl, pigeon,  
and other domestic birds.

(Fam. 2.) *Bothriocephalidæ*, or *Pit-headed Tape-Worms*.

*Bothriocephalus latus*: intestines of dog.

*Bothriocephalus cordatus*,

*Bothriocephalus fuscus*,

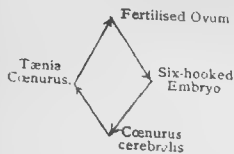
*Bothriocephalus reticulatus*,

*Bothriocephalus dubius*,

} Not recognised in this country.



FIG. 1.—LIFE HISTORY.



TÆNIA CŒNURUS

FIG. 2.



Ovum, with Six-hooked Embryo.

Segment X 6

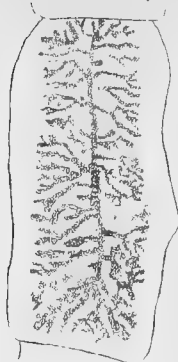


FIG. 3.

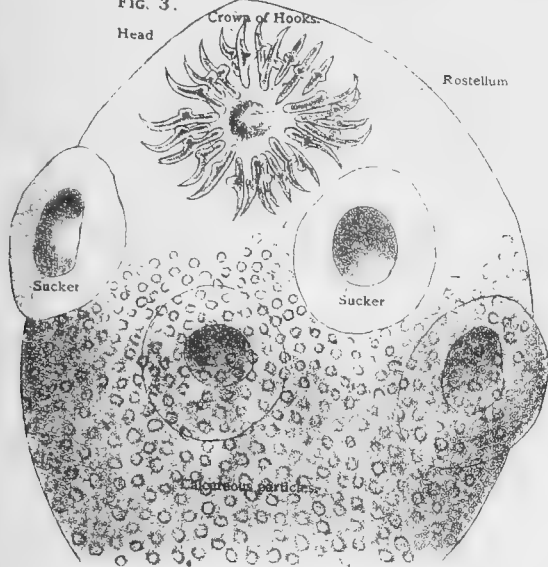
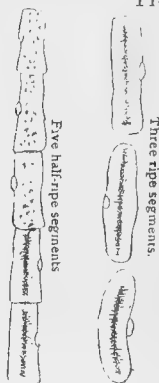
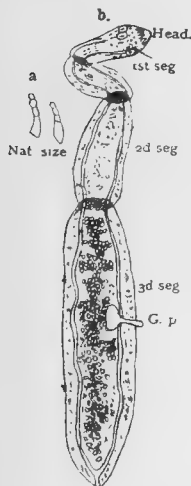


FIG. 4.



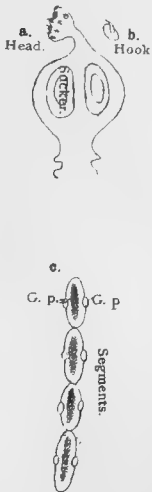
TÆNIA ECHINOCOCCUS.

FIG. 5.



TÆNIA CUCUMERINA.

FIG. 6



TÆNIA MARGINATA

FIG. 7.

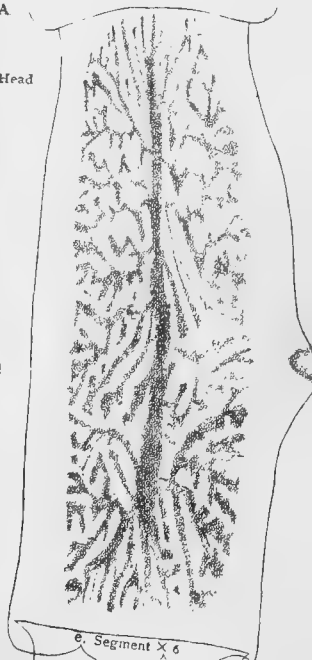
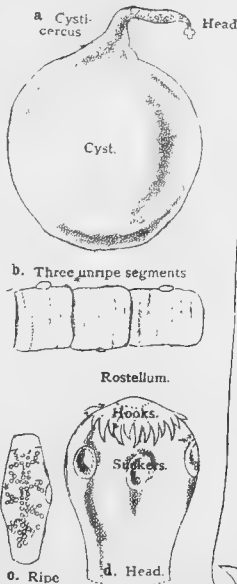




FIG 1. T. SERRATA.

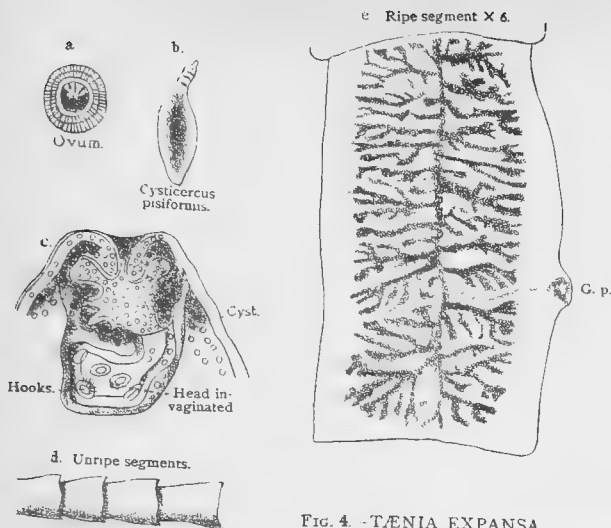


FIG. 2.—TÆNIA SOLIUM

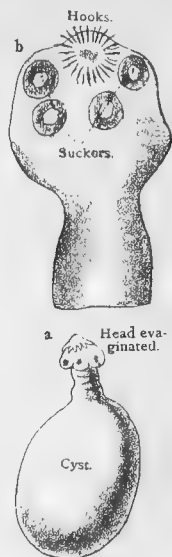


FIG. 4. -TÆNIA EXPANSA.

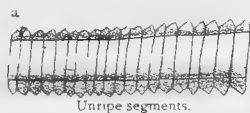


FIG. 5. TÆNIA GRASSICOLLIS. Segment X 6

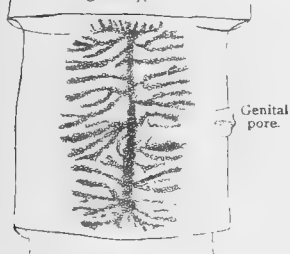


FIG. 3.—TÆNIA PERFOLIATA

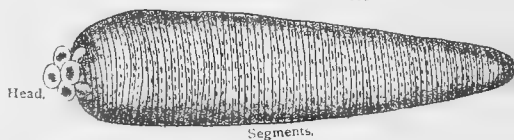


FIG. 7.

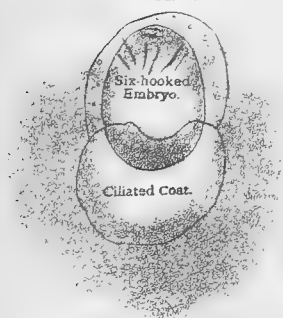


FIG 6.

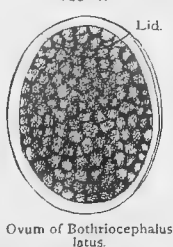


FIG. 8.

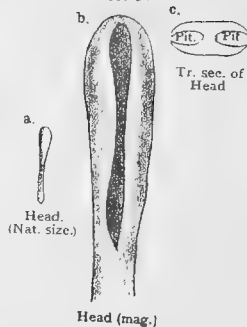






FIG. 3.  
ASCARIS LUMBRICOIDES.  
(Dissected)

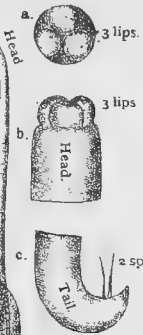


FIG. 4.—HEAD AND TAIL OF  
A. LUMBRICOIDES (Male).



FIG. 6.

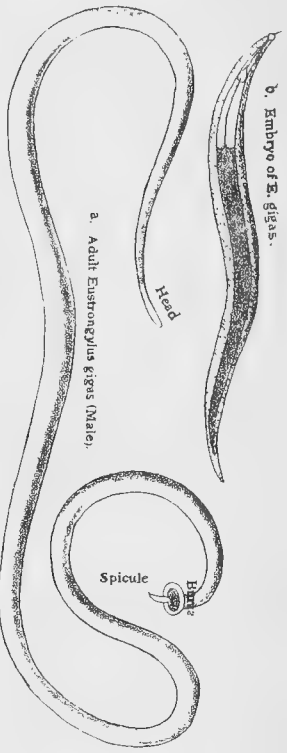


FIG. 8.  
OXYURIS VERMICULARIS.



FIG. 7.  
ASCARIS MYSTAX.

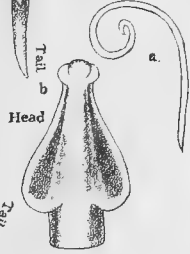


FIG. 11.

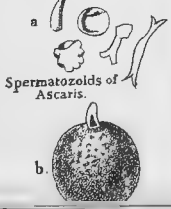


FIG. 9.  
DEVELOPMENT OF  
O. VERMICULARIS.

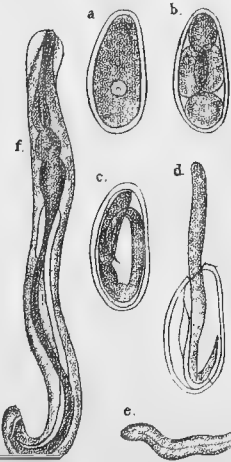


FIG. 10.

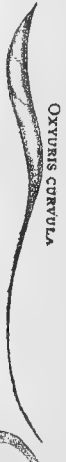




FIG. 1.—*FILARIA PAPILLOSA*.  
(Female.)

FIG. 2.  
*F. PAPILLOSA*.  
(Male.)

FIG. 4.  
TAIL END OF  
*F. PAPILLOSA*.

FIG. 3.  
HEAD OF *F. PAPILLOSA*.

FIG. 10.  
DISSECTIONS OF  
*S. ARMATUS*.

FIG. 7.  
HEAD OF *S*.  
*FILARIA*.

FIG. 5.

FIG. 6.

*STRONGYLUS FILARIA* (Female)

*STRONGYLUS FILARIA* (Male)

FIG. 8.  
TAIL OF *S*.  
*FILARIA*.

FIG. 11.  
HEAD OF *S. ARMATUS*.  
Teeth.

Bursa.  
Spicules.

FIG. 9.  
*STRONGYLUS ARMATUS*.  
(Nat. size.)

Act of Copulation.  
Sexually mature.  
Male.  
Female.

FIG. 13.  
*TRICHINA SPIRALIS* (Male).

FIG. 12.  
BURSA OF *S*.  
*ARMATUS*.

Head.

FIG. 14.  
ANTERIOR PART  
OF *T. SPIRALIS*.  
(Female.)

FIG. 15.  
*MUSCLE TRICHINÆ*.  
Nat. size.

Young *Trichina*.

FIG. 16.  
*MUSCLE TRICHINÆ*.  
(Mag.)

Two conical  
appendages.  
Cloaca.

FIG. 17.—ENCYSTED MUSCLE *TRICHINA*  $\times 80$ .

FIG. 18.—*TRICHOCEPALUS AFFINIS*.

a Female.

b Male





FIG. 1.—LIFE HISTORY.

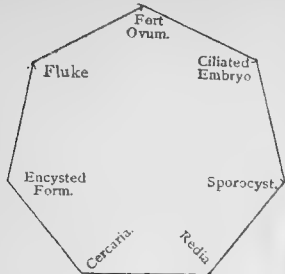


FIG. 2. EGG.



FIG. 4. SPOROCTYST.



YOUNG CERCARIAE.

FIG. 7. DISTOMUM LANCEOLATUM.

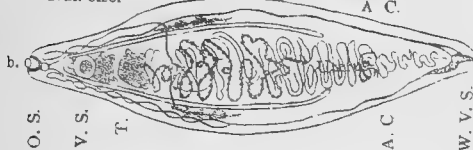


FIG. 3. CILIATED EMBRYO.

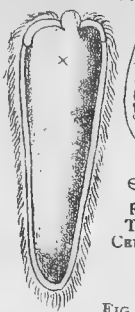


FIG. 5. TAILED CERCARIA.



FIG. 6.



DISTOMUM HEPATICUM.

FIG. 8. AMPHISTOMUM CONICUM.

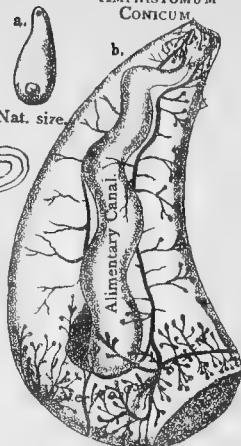


FIG. 9.—GASTROPHILUS EQUI

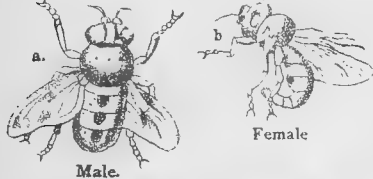


FIG. 10.—LARVA OF G. EQUI.



FIG. 11.—BURST PUPA OF G. EQUI.



FIG. 12.—PENTASTOMUM TENIOIDES. (Female.)

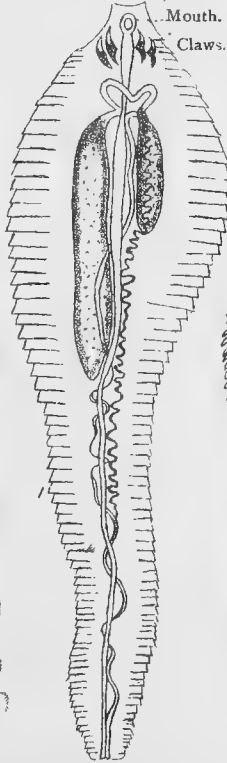


FIG. 14.—PENTASTOMUM DENTICULATUM.

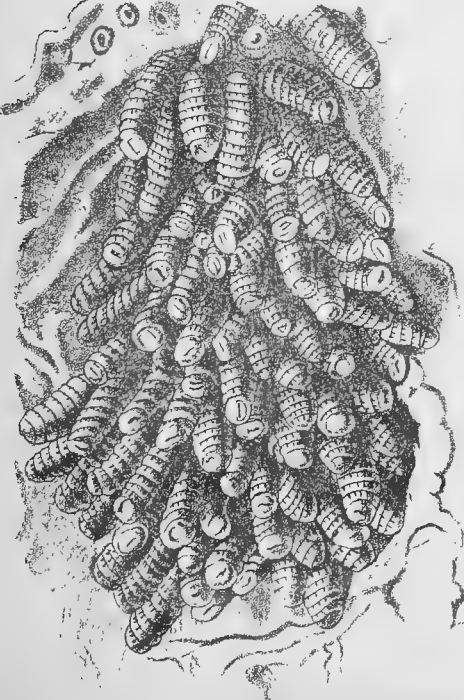
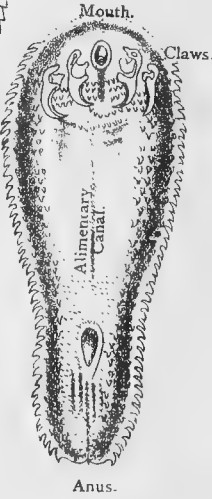






FIG. 1.  
Strongylus Refuseens in alveoli of Sheep's Lungs.  
( $\times 200$ .)

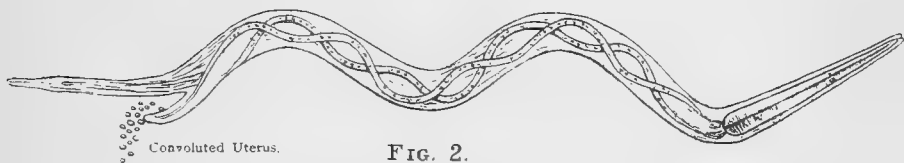


FIG. 2.  
Strongylus Contortus.



FIG. 3.  
Strongylus Contortus.—Bursa of Male.



FIG. 4.  
Strongylus Contortus.—Genital papillæ of Female.  
( $\times 50$ .)



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*For detailed and consecutive information the reader is referred to the Special Index on page 979.*

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